TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

EIGHTH SESSION

HELD IN MONTREAL, CANADA, MAY 25, 26, AND 27, 1896.

EDITED BY

FLOYD M. CRANDALL, M.D.

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CONTENTS.

PRESIDENTS.

1889. A. Jacobi, M.D.
1890. J. Lewis Smith, M.D.
1891. T. M. Rotch, M.D.
1892. Wm. Osler, M.D.
1893. A. D. Blackader, M.D.
1894. John M. Keating, M.D.
1895. F. Forchheimer, M.D.
1896. Joseph O'Dwyer, M.D.
1897. Samuel S. Adams, M.D.

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T. M. Rotch,
J. P. Crozer Griffith,
B. K. Rachford,
A. D. Blackader.

MEETING PLACES.

1888. Washington (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22 to 25.
1892. Boston, May 2, 3, and 4.
1893. West Point, May 24, 25, and 26.
1894. Washington, May 29 to June 1.
1895. Virginia Hot Springs, May 27, 28, and 29.
1896. Montreal, May 25, 26, and 27.

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ORGANIZATION OF THE AMERICAN PEDIATRIC SOCIETY.

After the adjournment of the Pediatric Section of the Ninth International Medical Congress, September 9, 1887, a meeting was held by a few of the members of the section, at which it was decided to organize an American Pediatric Society. Dr. J. Lewis Smith was elected temporary chairman and Dr. W. D. Booker, temporary secretary. The chairman was authorized to take measures to secure the co-operation of some of those physicians who took special interest in the advancement of the study of the diseases of children. The response to the invitations sent to such physicians showed a general desire for the organization of the Society. It was decided to leave the permanent organization to be effected by those who should participate in it. An announcement was, therefore, sent of a meeting to be held in September of the following year for the purpose of organizing the Society.

The American Pediatric Society met for permanent organization in Washington, D. C., September 18, 1888, in a parlor of the Arlington Hotel. In the absence of the temporary chairman, Dr. A. Jacobi was elected to that position. Drs. Jacobi, Booker, and Watson, were appointed a committee to draft a constitution. The committee reported the draft of a constitution which was unanimously adopted, after which the following permanent officers were elected: President, A. Jacobi, M.D.; First Vice-President, A. V. Meigs, M.D.; Second Vice-President, F. Forchheimer, M.D.; Secretary, W. D. Booker, M.D., Recorder, William Perry Watson, M.D.; Treasurer, Charles Warrington Earle, M.D.

It was moved that the Society apply for admission to the Congress of American Physicians and Surgeons. After electing Dr. A. Jacobi delegate to the Congress, the Society adjourned to hold its first annual meeting in September, 1889.

MINUTES OF THE EIGHTH ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY,

Held at Montreal, Canada, May 25, 26, and 27 1896.

First Day—*Morning Session.*

The First Vice-President, Dr. James C. Wilson, called the meeting to order in the meeting room of the Windsor Hotel at 11.15 A.M. The following members were present:—G. N. Acker, M.D., Washington; S. S. Adams, M.D., Washington; A. D. Blackader, M.D., Montreal; W. D. Booker, M.D., Baltimore; D. Brown, M.D., New York; A. Caillé, M.D., New York; W. S. Christopher, M.D., Chicago; F. M. Crandall, M.D., New York; J. Dorning, M.D., New York; R. G. Freeman, M.D., New York; J. H. Fruitnight, M.D., New York; J. P. C. Griffith, M.D., Philadelphia; L. E. Holt, M.D., New York; H. Jackson, M.D., Boston; C. G. Jennings, M.D., Detroit; H. Lafleur, M.D., Montreal; W. P. Northrup, M.D., New York; W. Osler, M.D., Baltimore; F. A. Packard, M.D., Philadelphia; C. P. Putnam, M.D., Boston; T. M. Rotch, M.D., Boston; A. Seibert, M.D., New York; C. W. Townsend, M.D., Boston; A. H. Wentworth, M.D., Boston; J. C. Wilson, M.D., Philadelphia; L. M. Yale, M.D., New York.

In the absence of the President, Dr. Joseph O'Dwyer, of New York, the President's Annual Address entitled "The Evolution of Intubation," was read by Dr. W. P. Northrup, of New York.

Dr. George N. Acker, Washington, read a paper on "Gangrene of the Lung Complicating Typhoid Fever."

Dr. J. Henry Fruitnight, New York, read a paper entitled "Malignant Endocarditis," and presented a specimen.

Discussion by Drs. Northrup, Holt, and Osler.

Dr. Floyd M. Crandall, New York, read a paper on "The Occurrence of Influenza in Children, with a Report of Local Epidemics."

Discussion by Dr. Seibert.

Dr. Irving M. Snow, Buffalo, read a paper entitled "Papilloma of the Larynx in an Infant Aged One Year."

Dr. B. K. Rachford, Cincinnati, read by title a paper on "Superficial Gangrene."

Dr. F. Forchheimer, Cincinnati, read by title a paper on "Elimination an Etiological Factor of Diseases of the Alimentary Canal."

Evening Session.

Dr. Augustus Caillé, New York, read a paper entitled "Local Treatment for Tubercular Meningitis."

Dr. Arthur Howard Wentworth, Boston, read a paper entitled "Some Experimental Work on Lumbar Puncture of the Sub-arachnoid Space."

Dr. Charles G. Jennings, Detroit, read a paper entitled "Lumbar Puncture of the Sub-arachnoid Space."

Discussion on Lumbar Puncture, by Drs. Holt, Blackader, Caillé, Wentworth, Fruitnight, and Jennings.

Dr. Samuel S. Adams, Washington, reported a case of "Temporary Insanity Following Typhoid Fever."

Discussion by Drs. Northrup, Crandall, Wilson, Osler, Christopher, and Wentworth.

Dr. Frederick A. Packard, Philadelphia, read a paper entitled "Endothelioma of the Brain, with Atrophy of Paralyzed Members."

Dr. Henry Jackson, Boston, read a paper on "Nasal Feeding in Diphtheria."

Discussion by Drs. Holt, Fruitnight, Jennings, and Wentworth.

Dr. William Osler, Baltimore, read a paper on the "Classification of the Tics or Habit Movements."

Discussion by Drs. Christopher, Osler, and Fruitnight.

Second Day—*Morning Session.*

On motion of Dr. Northrup the society went into executive session to consider the subject of publication of the transactions.

Upon adjournment of the executive session the "Report of the Committee upon Collective Investigation of the Antitoxin Treatment of Diphtheria in Private Practice" was read by the chairman, Dr. L. Emmett Holt.

Dr. Samuel S. Adams, Washington, read a paper on "Comparative Results of the Treatment of Diphtheria with and without its Antitoxin in the District of Columbia."

Dr. Frederick A. Packard, Philadelphia, read a paper on "Favorable Results of Diphtheria Antitoxin Treatment."

Dr. A. Seibert, New York, read a paper on "Sudden Death after Antitoxin Injections."

A general discussion on the antitoxin treatment of diphtheria was participated in by Drs. Rotch, Caillé, Northrup, Seibert, Osler, Lafleur, Holt, Wilson, Yale, Dorning, Adams, Booker, Townsend, Crandall, Fruitnight, Wentworth, and Griffith.

Afternoon Session.

Dr. Rowland G. Freeman, New York, read a paper entitled, "Is Low Temperature Pasteurization of Milk, at about 67° C., Efficient?"

Discussion by Drs. Rotch, Northrup, Holt, and Griffith.

Dr. Henry D. Chapin, New York, read by title a paper entitled a "Study of Premature Infants Treated in the Incubator."

Dr. Charles W. Townsend, Boston, read a paper on "Thigh-friction in Infants under One Year."

Discussion by Drs. Adams, Yale, Caillé, Holt, Booker, Griffith, and Wentworth.

Dr. William P. Northrup, New York, reported a case of "Apparently Relapsing Cerebro-Spinal Meningitis, Followed by Death and Autopsy."

Discussion by Drs. Townsend, Osler, Wentworth, Holt, Christopher, Caillé, and Rotch.

Dr. Henry Lafleur, Montreal, presented notes on a "Case of Insolation in an Infant Aged Thirteen Months."

Discussion by Drs. Fruitnight, Seibert, Griffith, Adams, and Wentworth.

Dr. A. D. Blackader, Montreal, reported a case of "Enlargement of the Liver in a Child, with Symptoms closely resembling Typhoid Fever."

Discussion by Drs. Caillé, Lafleur, Seibert, and Booker.

Evening Session.

Executive Session.—The report of the Council was submitted, and the following officers were elected for the coming year.

President, Samuel S. Adams, M.D., Washington, D. C.
First Vice-President, W. S. Christopher, M.D., Chicago.
Second Vice-President, Charles P. Putnam, M.D., Boston.
Secretary, Frederick A. Packard, M. D., Philadelphia.
Treasurer, Charles W. Townsend, M.D., Boston.
Recorder, Floyd M. Crandall, M.D., New York.
Member of Council, William Osler, M.D., Baltimore.

The following were elected members: Dr. John H. Musser,

Philadelphia; Dr. Charles G. Kerley, New York; Dr. J. Lovett Morse, Boston, and Dr. Thompson S. Westcott, Philadelphia.

During the first executive session the following proposition was submitted by the editor of the ARCHIVES OF PEDIATRICS:

TO THE COUNCIL, AMERICAN PEDIATRIC SOCIETY:

GENTLEMEN: In regard to the publication of the transactions of this Society, I beg to say that I will publish the papers read at the various meetings, in the ARCHIVES OF PEDIATRICS as rapidly as possible and will furnish the Society two hundred bound copies of transactions when the publication of the papers has been completed, not furnishing reprints to authors gratuitously.

Authors who publish their papers in other journals will be expected to bear the expense of the reproduction of said papers in the Transactions, such expense to be only the actual cost of type-setting and printing.

I will not consent to publish symcronously with other journals papers which have already appeared in abstract.

I reserve the right to decline to publish in the ARCHIVES OF PEDIATRICS any paper which has been so fully reproduced in abstract in one or more other journals as to render it in my opinion of no value to the ARCHIVES.

Yours truly,

(Signed) FLOYD M. CRANDALL.

After discussion the above proposition was accepted by vote of the Society.

The following resolution was presented by the Council:

Resolved, That no reports of articles read before the Society be published, except those taken by the official stenographer, and then only after having been approved by the Council.

After discussion this resolution was adopted.

On motion the Committee on Antitoxin was continued for another year and the sincere thanks of the Society were extended for the excellence of the report presented.

Drs. Rotch, Caillé, and Northrup were appointed a Committee to formulate resolutions expressing the conclusions of the Society regarding the use of antitoxin. These resolutions were reported at the final session and the editor was directed to append them to the antitoxin report.

Resolutions on the Anti-Vivisection Act for the District of

Columbia, similar to those adopted by the American Medical Association were adopted by the Society.

The report of the Treasurer showing a deficit of about forty dollars was submitted. After being audited and reported correct, it was accepted by the Society.

On motion the Council was impowered to appropriate a sum not to exceed fifty dollars for the improvement of the Transactions.

After the adjournment of the executive session, the members of the Society were received by Dr. and Mrs. Blackader, of Montreal.

Third Day—*Morning Session.*

This session was devoted to the presentation of pathological specimens. Specimens were presented as follows:

Dr. T. M. Rotch—An Unusual Form of Congenital Cardiac Malformation.

Dr. Joseph O'Dwyer—Croup Laryngis.

Dr. L. Emmett Holt—(a) Perforative Appendicitis in a Child two and one-half years of age. (b) Adhesive Pericarditis, with entire obliteration of the pericardial sac, in a child aged sixteen months.

Discussion by Drs. Rotch. and Osler.

Dr. Augustus Caillé—(a) Cicatricial Stenosis of the Larynx. (b) Congenital Heart Lesion.

Discussion by Drs. Northrup, and Caillé.

Dr. Samuel S. Adams—(a) Abscess of the Brain. (b) Congenital Pharyngo-Oesophageal Stenosis.

Dr. Frederick A. Packard—Congenital Heart Disease.

Dr. George N. Acker—(a) Organic Disease of the Heart, following scarlatina. (b) Sarcoma of Thymus and Bronchial Glands.

Dr. Henry D. Chapin—A Case of Tumor of the Brain.

Dr. Rowland G. Freeman—Adherent Pericardium (two cases).

Dr. J. P. Crozer Griffith—Perforate Septum Ventriculorum.

Discussion by Drs. Rotch, Osler, Caillé, and Wentworth.

Resolutions expressing the sincere regret of the Society at the necessary absence of Dr. O'Dwyer were adopted.

The thanks of the Society were extended to Dr. Blackader

and to the medical profession of Montreal for the many courtesies received.

The thanks of the Society were also extended to Dr. H. Seward Webb for courtesies extended by him.

After a unanimous vote of thanks to the acting President, Dr. James C. Wilson, the meeting adjourned.

FLOYD M. CRANDALL, M.D.,
Recorder.

JOSEPH O'DWYER, M.D.

THE EVOLUTION OF INTUBATION.

The Annual Address of the President of the American Pediatric Society.*

BY JOSEPH O'DWYER, M.D.

New York.

I have been requested by several members of this Society to make the leading steps in the evolution of intubation the subject of the presidential address. I have consented to do this for two reasons. First, because I wish to demonstrate that the development of this procedure from the beginning to the end was accomplished without borrowed inspiration. Second, because I desire to call special attention to the great importance of systematic thinking, of the value of which I was totally ignorant prior to the beginning of these experiments. I have repeatedly verified the truth of the saying of one of the philosophers, that by persistently thinking a subject over a flood of light is thrown upon it; the light at first comes slowly but constantly increases with practice. As George Elliot aptly puts it, "Growing thought makes growing revelation." The tedious, laborious efforts necessary to beget a new idea in the early stage of these experiments, compared with the ease and rapidity with which they came at a later period, when the habit of thinking was well established, was indeed a revelation to me and I am sure would be to any one of ordinary intelligence who would take the trouble to cultivate the habit. My reason for emphasizing this matter is the firm conviction that very few have any clearer idea of its importance than I had until the necessity for thinking outside of the beaten track was forced upon me.

What led to the first intubation experiment? is the question

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* Eighth Annual Meeting, Montreal, May 25, 1896.

I have been asked more frequently than any other. Complete failure with tracheotomy in the New York Foundling Hospital extending over a period of several years, was the real incentive to the work. From the foundation of this institution in 1869 to the inception of these experiments in 1880 we could not point to a single recovery following tracheotomy, to offset the prejudice that existed against it. Dante's inscription over the portals of the infernal regions "Hope abandon all who enter here" might well have been placed over the door of the croup room in the Foundling Hospital. All that we could say in favor of the operation was that it allowed the little sufferers to die easier. It was a justifiable means of euthanasia—not a very powerful argument with the laity, when the means of attaining it are considered. The operation finally came into such bad odor that in the service of some of my colleagues it was not resorted to at all. The resident physician would ask, "What will I do if the little patient gets worse in the night?" Continue the spray, or whatever treatment was in vogue at the time, would be the answer. Such was the condition of things just prior to the conception of intubation and, as I have already stated, was the parent of it.

Without attempting to explain the cause of such ignominious failure with tracheotomy it is important to state that the number of croup cases treated during any one year was not very large because they were drawn only from the children connected with the institution, consisting of six or seven hundred resident and about twelve hundred who were wetnursed or otherwise cared for outside. During the year 1879 some modifications of the tracheal canula were made for the purpose of increasing the expulsive power of the cough. It was thought that if the poisonous secretions could be more effectually gotten rid of, the secondary pneumonia so frequently the cause of death might be obviated or at least diminished. After a trial in several cases these modifications proved to be useless.

The possibility of overcoming obstruction to respiration through the natural passage has probably occurred to almost every tyro in medicine, when confronted with his first case of croup. Why not try a catheter passed through the mouth or nose, is a question that I had often heard in my early practice. Such was the beginning of intubation. Various kinds of catheters were used and passed into the larynx through the nose because they could not be retained long in the mouth. Besides

the difficulty of introduction and the great irritation produced, such a long channel could not be kept clear, and this method of overcoming the obstruction was soon demonstrated to be impracticable, but it served the important purpose of suggesting something else. It soon occurred to me that the channel for the passage of air and secretions might be made very short by a tube constructed so that the proximal end would rest solely in the larynx and thus allow the epiglottis to close over it during the act of swallowing. When this was determined upon the first occasion for serious thinking presented itself. What form of tube could be constructed that would be retained in the larynx against the expulsive power of coughing and at the same time not endanger the integrity of the inflamed and swollen tissue by undue pressure? The tracheal canula is held in position by a tape securely tied around the neck. What device applied to the laryngeal tube would take the place of this tape? This was the first question to be answered. After considerable thought on the subject, nothing occurred to me that would fulfil these indications except a tube constructed on the bivalve principle, the blades of which would be closed during insertion and opened by means of a spring when detached from the introducer. The first difficulty encountered with this form of tube was in regulating the strength of the spring. If too weak, the blades, when set free, failed to separate and apnœa resulted; if too strong, ulceration was produced around the distral extremity from the continuous pressure. But the greatest difficulty and the one that proved insurmountable was to get rid of the open spaces between the blades when *in situ*. Into these spaces the swollen mucous membrane gradually intruded, obliterating the breathing space at first obtained. These tubes were always retained and usually gave prompt relief to the dyspnœa, which sometimes lasted for several days. Secondary tracheotomy was performed in all the most hopeful cases, and one recovered after wearing the canula six months. Twice the larynx was laid open before the canula was finally gotten rid of, but the voice was permanently impaired. This occurred in November, 1882, and was the first operated case of croup that recovered in the Foundling Hospital since its foundation in 1869, a period of over thirteen years. The shoulder on the bivalve tubes was made with a solution of gutta percha in chloroform, applied layer after layer until a sufficient thickness was obtained. After about three years' experi-

ments with the bivalve it was reluctantly given up as useless.

About this time I was informed of Bonchut's experiments with tubage, and the temptation to look up the literature and learn what kind of tubes he had tried was very great. I was also told by some of my friends that I could write a very interesting paper on the work already done, but I was convinced that yielding to either of these temptations would be the end of intubation.

Soon after this the croup cases occurring amongst the out-children were not returned to the hospital for treatment. The belief was prevalent that these children were being subjected to unnecessary suffering without any tangible result, as if any suffering could be greater than that of slow strangulation. Tracheotomy was bad, but intubation so far was worse because it interfered so seriously with feeding. An impassable barrier was therefore established between the out-door department and the much-dreaded croup room. If by any chance a case should get in, it was hurried back to its unsavory tenement home where, surrounded by every unsanitary condition to be found among New York's poorest foreign population, whence the best wet-nurses are derived, its chances of life were considered better than if admitted to the abode of cleanliness, skillful nursing and more than mother's care. The experimental work was, therefore, restricted to the croup developing among the resident children and was consequently somewhat retarded.

After some time I concluded to try a tube of plain oval form, about one inch in length, with a small slit in the posterior portion of the upper extremity for insertion of the extractor. At this point there is a break in my records extending from August, 1883, to April, 1884, which I cannot account for.

The first case of croup that developed after the new tubes were obtained was in an infant aged two months and twenty-four days. The smallest size was inserted, and gave complete relief, and to my surprise was retained until the child died sixteen hours later, free from any return of dyspnœa. I show you the tube used in that case. The second case was a girl aged four years, and was intubated May 21, 1884. Here also the tube was retained, and was removed in sixty-seven hours, but had to be replaced in five hours. While attempting to reinsert the tube he little patient closed her teeth firmly on the metallic shield

protecting the finger in lieu of mouth gag, and refused to let go. I could neither advance nor recede, and was obliged to administer chloroform to complete the operation. The tube was coughed out in three days, and was not again needed. This was the first recovery in the history of intubation, and was, therefore, a very important event. The question of permanent injury to the vocal cords, the one thing dreaded more than any other, was now to be settled, and the return of the voice was therefore watched with considerable anxiety. It did not return for several weeks, but the cough continued to have a croupy character for some time after the tube was dispensed with, which was regarded as a good omen, because it was believed that if the cords were even partially destroyed the cough would be toneless. This case also demonstrated the necessity of a mouth gag, which was devised soon afterwards. Seven more cases were treated with this form of tube, and all were fatal.

The mucous membrane crowded through the little buttonhole in the rear, and served as the starting point for the accumulation of secretions, and it was therefore dispensed with. Tubes, the same in every other particular except the opening, were constructed, which necessitated a new form of extractor, and the one I show you was devised. These tubes were not retained at all unless a very large size was used, which proved that the preceding variety was held in position by the mucous membrane buttoning into the opening left for the extractor. These tubes were used only in five cases, one of which recovered in December, 1884, after wearing the tube ten days. It was repeatedly expelled, and several times a fatal asphyxia was averted only at the last moment. Intubation was now looking up. It had two recoveries to its credit in five years, and the prejudice against it was consequently abating. All the tubes so far used were very short, the longest being only a little over an inch. The necessity for longer ones had been demonstrated several times by finding obstruction around the distal extremity after death. It was decided to use tubes long enough to reach clear to the bifurcation, for the double purpose of overcoming obstruction in the trachea and testing their retaining qualities. A number of measurements of the trachea of different ages were first made, and the tubes were constructed accordingly, the smallest being one and three-fourths inches and the longest three inches long.

These tubes were never expelled, but would be projected up-

ward by every spell of coughing, and would stand in that position until pushed down with the finger. This was some improvement on the preceding variety, because the tube on account of its length, could not leave the larynx. Even if the proximal end were in contact with the vault of the pharynx, the distal end was still below the vocal cords, but there never was any obstruction under these circumstances, as would naturally be expected. After coughing, and especially before feeding, the nurse was directed to inspect the throat, and if the tube was standing up, to push it down with her finger. These tubes were used in only six cases, all fatal. The first case on which they were tried demonstrated that the solution of the problem was not yet reached, and I only continued to use them until I had time to think of something better. This came in the form of a second shoulder about half an inch below the first, which left a furrow for the vocal cords to rest in. To avoid any interference with insertion, this lower shoulder was wedge-shaped, with the thick end looking upward. There was no question about the retaining qualities of this device. In the first case in which it was tried the weak extractor then used failed to budge it from its position after several attempts, and as the temperature at this time was 105.5°, respiration 90, and the pulse could not be counted, it was not necessary to do anything else.

The next step was to reduce the size of the retaining shoulder in order to lessen the grip of the vocal cords. When this was done, they were not held down in the larynx much better than the long oval tubes. These were used in ten cases, of which one recovered, making the third recovery since the beginning of the experiments.

It was evident from the start that this device for retaining the tubes in the larynx would never prove satisfactory, and I began almost immediately to cast around for something better. I had now learned how to think, and in a little over one month from the time the first shoulder was tried I thought of the very simple and effectual expansion in the middle of the tube now known as the retaining swell. That this was the solution of the most difficult part of the problem connected with intubation was apparent almost without a trial. This device would not interfere either with introduction or removal, as the thick portion of the tube would rest well within the trachea, where there was ample room, and therefore no danger from pressure. It required a large

number of experiments to determine the proper size for the retaining swell, so that the tubes would be retained, and at the same time not hold too firmly. It was hoped that this could be so regulated as to retain a tube as long as required, and allow of its expulsion when occluded, but this was found impossible because of the great differences in the size of larynges at the same age.

While these experiments were progressing other improvements suggested by the *post-mortem* findings were being worked out. In the *post-mortem* records constant references were made to the presence of ulceration at three principal points. First, in the cricoid division of the larynx just below the vocal cords. Second, at the base of the epiglottis from pressure during the act of swallowing. Third, on the anterior wall of the trachea at the distal extremity of the tube. The subglottic ulceration extending through the whole thickness of the mucous membrane soon demonstrated that the tubes first employed were far too large and that it was the lumen of this portion of the larynx and not that of the chink of the glottis or trachea that must determine the size of the intubation tube. If respiration could not be carried on through a much smaller space, intubation would not be possible. The cutting down process was therefore begun very early, as you will see by comparing some of the samples that I will pass around. When ulceration ceased to occur at this point there was still ample breathing space left. Attempts to get rid of the ulceration at the base of the epiglottis were also made early. This was accomplished by giving the upper portion of the tube a backward curve leaving the metal on the anterior surface thick enough so that a blunt rounded surface met the epiglottis when the latter was pressed upon it by the base of the tongue during deglutition. The head was also gradually increased in size which contributed to the same result by distributing the pressure over a larger surface.

The ulceration on the anterior wall of the trachea which often laid bare the cartilages was a more difficult matter to deal with. It was not understood for some time how this ulceration, which extended at least a quarter of an inch above and below the distal extremity of the tube, was produced. The injury below was produced by the mucous membrane being drawn upwards in contact with the sharp edge of the tube in the act of swallowing, while that above occurred in the downward move-

ment as the trachea returned to its normal position. The head of the tube being fixed in the larynx, its distal extremity, therefore, moved over a space of about half an inch during every act of swallowing. This defect was remedied by leaving the metal at this point even thicker than above and rounding it off so as to present a smooth blunt surface that would slide up and down over the tissues without injury.

The tubes were now practically complete but the extractor was very imperfect and my attention was next directed to the improvement of this instrument, which was accomplished during the summer of 1886. Much fault has been found with this extractor and many are the attempts that have been made to improve it, but without success, because it is not susceptible of improvement unless as a member of this Society aptly put it, one can be devised that will find the hole. It is the difficulty of finding the little hole in the tube that necessitates finding fault with the instrument; because the operator cannot afford to be at fault. It is possible that the electro-magnet may yet be so perfected as to render this part of the operation less difficult.

The only impediment to the general adoption of intubation that remained was the difficulty of feeding the intubated patient. As you will have noticed, the heads of all these experimental tubes were left very small with the object of allowing them to sink very low in the larynx and thus avoid interference with swallowing so far as possible. While gradually increasing the size of the heads to their present dimensions no perceptible difference could be discovered in the ability to swallow. Various modifications in the shape of the head were next tried, such as having it higher behind than in front so as to meet the epiglottis half way, then reversing this and making the oblique from before, backwards and downward; making the head concave from side to side and various other devices were tried even to an artificial epiglottis.

After repeated and long continued experiments, it was demonstrated that no artificial device could overcome the difficulty of swallowing, while the constriction of the larynx is prevented by the presence of the tube. It was also shown that when a tube is worn in the larynx for several weeks the ability to swallow perfectly is acquired. The explanation of this is found in the fact that this vital passage is doubly guarded against the entrance of foreign matter by the epiglottis and the constrictor

muscles. It is difficult to say which is the more important. When either is destroyed the other soon learns to assume the duties of both. Many cases are on record in which total destruction of the epiglottis was followed by only temporary interference with deglutition, and on the other hand numerous examples have been observed in which the ability to swallow perfectly with a tube in the larynx has been acquired. In the ordinary run of croup cases the tube is not worn long enough for this purpose.

The greatest danger attending the wearing of an intubation-tube is the obstruction of its distal extremity by a cast of false membrane suspended from the larynx, where it remains long adherent but completely detached below. The next experiments were therefore directed to getting rid of such loose membrane when discovered. Various devices were tried, some of which I will show you. The first trial was with a bivalve wire spring long enough to reach to the bifurcation. It was introduced closed and when down the spring was released and the blades scraped the sides of the trachea as it was withdrawn. It was useless. The next device consisted of a long tube made of soft silver, perforated at its distal extremity, where it was surrounded by thin rubber which could be quickly inflated by a small rubber bulb fixed in the handle. This was tried only once. It was passed well down to the bifurcation, the bulb in the handle compressed, and, as it was being withdrawn, there was an explosion audible at a considerable distance. The balloon had burst in the trachea. A metallic instrument constructed on the same principle was then tried. It was introduced closed and the extent to which it would open could be graduated according to the size of the trachea before insertion. This also proved useless. Another device intended to break up the membrane so that it could be expelled piece by piece also proved ineffectual.

It was this idea of breaking up the membrane that suggested the larger caliber tube. A cast of the trachea is too large to pass either through the small lumen of the intubation-tube or the constricted chink of the glottis, unless it follow the tube immediately or before the stenosis recurs. More room, therefore, was what was needed, and the short cylindrical tubes gave three or four times the caliber of the ordinary croup tubes. They are constructed of very thin metal and as large as the cricoid division of the larynx will admit by using some force, else they

will not be retained, as the retaining power is lateral pressure only. They are never to be left in position longer than a few hours at a time, as the pressure would soon destroy the mucous membrane lining, the cricoid cartilage and the cartilage also. These tubes have proved fairly successful in getting rid of false membrane when loose and not anchored below by extension into the bronchi. Even under the latter circumstances, it is not rare to have a cast of the trachea expelled with several branches attached, but only with temporary relief. The completion of these tubes practically terminated the experiments with the intubation instruments for children.

In December, 1885, before the croup tubes were perfected, a woman who was suffering from chronic syphilitic stenosis of the larynx was sent to me for treatment. The case was urgent, immediate tracheotomy having been advised, to which she would not consent. There were no adult tubes then in existence. In fact I had scarcely given the subject a thought. After a few hasty measurements of the normal adult larynx, I had several tubes constructed of different sizes, none of which could be forced through the stricture, and it was necessary to use the largest of the children's tubes, which gave complete relief. She complained so much of the irritation produced by the string that it was removed. The head of this tube was so small that nothing but a close cicatricial stricture prevented it from dropping into the trachea. It sank deeply in the larynx and was partially covered by a cicatricial band which rendered its removal very difficult, but it was accomplished by means of a forceps, as the children's extractor, then the only one in use, was not long enough to reach it. Other cases of chronic stenosis followed in quick succession. On the experience thus derived, the set of adult tubes was constructed. The retaining swell on these tubes is made comparatively small, in order to facilitate their passage through a stricture, graded on the same principle as urethral sounds. On this account they will not be well retained in acute stenosis of the larynx unless a pretty large size is used.

There is only one means of producing efficient and at the same time prolonged artificial respiration and that is the bellows method devised by George E. Fell of Buffalo. In order to simplify the connection between the bellows and the air passages, and to avoid the necessity of resorting to tracheotomy, I devised a set of laryngeal tubes which accomplish this object perfectly.

It was necessary that the glottis should be completely tamponned so that no air could return beside the tube and this was difficult to do in the vestibule of the larynx, because of its distensibility and still more so in the chink because of its triangular shape. The subglottic division of the larynx which is almost cylindrical, unyielding, and comparatively small was therefore selected as the part most easily tamponned. The great variety in size of adult larynges, which is even more marked than in children, rendered it necessary to make the distal ends of the tube conical in shape so that each one would suit several cases. The furrow that surrounded the portion that occupies the chink of the glottis are intended for the vocal cords which aid in retaining them.

A laryngeal tube with triangular opening in the side to act as a snare for the removal of subglottic growths was also devised and some work was done on a laryngeal speculum to facilitate the removal of neoplasms from other regions of the larynx, but these instruments were not perfected because I was not engaged in any throat work except that incidental to general practice.

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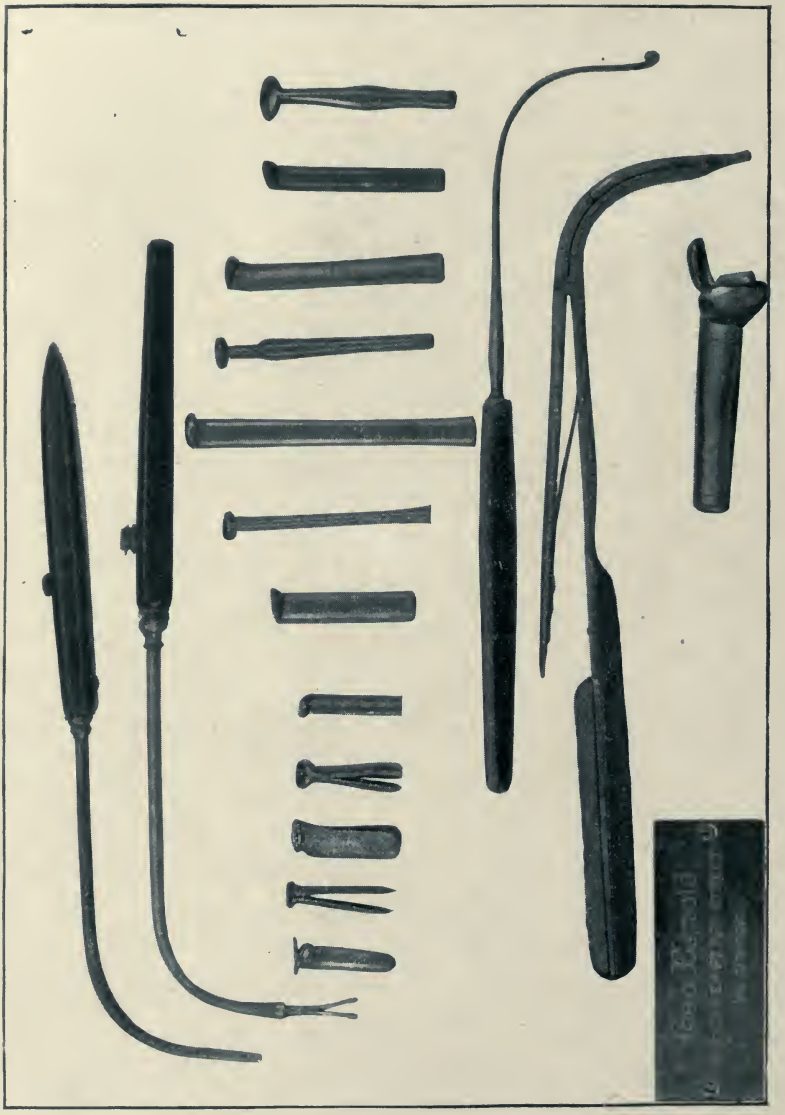

TUBES AND INSTRUMENTS ILLUSTRATING THE EVOLTUION OF INTUBATION.

THE REPORT OF THE AMERICAN PEDIATRIC SOCIETY'S COLLECTIVE INVESTIGATION INTO THE USE OF ANTITOXIN IN THE TREATMENT OF DIPHTHERIA IN PRIVATE PRACTICE.*

This subject was chosen by the officers of the Society for its eighth annual meeting, with the belief that a large amount of valuable experience not otherwise available, might in this way be reached and collated. It was also believed that a more trustworthy estimate of the value of the serum treatment of diphtheria might thus be obtained than by statistics taken from hospital practice. There are very few hospitals in America that receive diphtheria patients, and the conditions under which patients are admitted to hospitals and the surroundings while there, are so different from those of private practice, that the measure of success in hospital cases cannot be taken as an index of the results which have been obtained upon this side of the Atlantic with the new treatment.

In order, therefore, to obtain an expression of opinion from American physicians as to the serum treatment, after what had been, with most of them, their first year's experience, a circular letter was prepared and issued by the Committee early in April. This was distributed through the members of the Society as widely as could be done during the time allowed. An attempt was made to reach as many physicians as possible who had had experience with the remedy.

The first surprise of the Committee was in learning how very widely the serum treatment had been employed, especially in the Eastern and mid-Western states. With more time, the

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* Reported at the Eighth Annual Meeting held at Montreal, Canada, May 26, 1896.

number of cases collected might easily have been doubled and perhaps trebled; but enough reports have come in to enable one to see what opinion was held on the 1st of May, 1896, by American physicians who have used this remedy.

The circular letter asked for information upon the following points: Age; previous condition; duration of disease when the first injection was made; the number of injections; the extent of the membrane—tonsils, nose, pharynx, and larynx; whether or not the diagnosis was confirmed by culture; complications or sequelæ, viz., pneumonia, nephritis, sepsis, paralysis; the result; and remarks, including other treatment employed, the preparation of antitoxin used, and general impression drawn from the cases.

Reports were returned from 615 different physicians, with 3,628 cases. Of these, 244 cases have been excluded from our statistical tables. These were cases in which the disease was said to have been confined to the tonsils and the diagnosis not confirmed by culture, and therefore open to question. A few cases were reported in such doubtful terms as to leave the diagnosis uncertain. The figures herewith given are therefore made up from cases in which the diagnosis was confirmed by culture (embracing about two-thirds of the whole number) and others giving pretty clear evidence of diphtheria, either in the fact that they had been contracted from other undoubted cases, or where the membrane had invaded other parts besides the tonsils, such as the palate, pharynx, nose, or larynx. It is possible that among the latter we have admitted some streptococcus cases, but the number of such is certainly very small.

There are left then of these cases, 3,384 for analysis. These have been observed in the practice of 613 physicians from 114 cities and towns, in fifteen different states, the District of Columbia and the Dominion of Canada.

In the general opinion of the reporters the type of diphtheria during the past year has not differed materially from that seen in previous years, so that it has been average diphtheria which has been treated. If there is any difference in the severity of the cases included in these reports from those of average diphtheria, it is that they embrace a rather larger proportion of very bad cases than are usually brought together in statistics. The cases according to the extent of the membrane, are grouped as follows: In 593 the tonsils alone were involved. In 1,397 the tonsils and

pharynx, the tonsils and nose, the pharynx and nose, or all three were affected. In 1,256 cases the larynx was affected either alone or with the tonsils, pharynx, and nose, one or all. In many instances the statement is made by the reporters that the serum was resorted to only when the condition of the patient had become alarmingly worse under ordinary methods of treatment. This is shown by the unusually large number of cases in which injections were made late in the disease. Again, many physicians being as yet in some dread of the unfavorable effects of the serum have hesitated to use it in mild cases and have given it only in those which from the onset gave evidence of being of a severe type. The expense of the serum has unquestionably deterred many from employing it in mild cases. These facts, it is believed, will more than outweigh the bias of any antitoxin enthusiasts by including many mild cases which would have recovered under any treatment. It will, however, be remembered that tonsillar cases not confirmed by culture have not been included.

Only two reports embracing a series of over 100 cases have been received, most of the observers having sent in from five to twenty cases, although there are many reports of single cases, particularly of single fatal ones.

In addition to this material which has come in response to the circular, there have been placed at the disposal of the Committee by the courtesy of Dr. H. M. Biggs, 942 cases treated in their homes in the tenements of New York. Of these, 856 were injected by the corps of inspectors of the New York Health Board, upon the request of the attending physician, and eighty-six others were treated by physicians receiving free antitoxin from the Health Board. In the first group the diagnosis of diphtheria was confirmed by culture in every case, and in all of the latter except twenty-six; in these the diagnosis rested upon extensive membranous deposits or laryngeal invasion. The cases of the New York Health Board were of a more than ordinarily severe type, 485, or more than 50 per cent. of these being reported as being in bad condition at the time of injection; to mild cases the inspectors were not often called. Further, an unusually large number of them (38 per cent.) were injected on or after the fourth day of the disease. In 182 of these cases only the tonsils were affected; in 466 the tonsils with the pharynx or nose, the pharynx and nose, or all three; in 294 the larynx was

invaded either with or without disease of the tonsils, nose, or pharynx.

Through the courtesy of Dr. Biggs the Committee is able to include also a partial report upon 1,468 cases from Chicago, treated in their homes in that city by a corps of inspectors of the Health Department. It was the custom in Chicago to send an inspector to every tenement-house case reported, and to administer the serum unless it was refused by the parents. These cases were therefore treated much earlier and the results were correspondingly better than were obtained in New York, although the serum used was the same in both cities, viz., that of the New York Health Board.

THE RESULT AS INFLUENCED BY THE TIME OF INJECTION.

In Table I. are given the results obtained in these three different groups of cases, classified according to the day on which they received the first injection of serum antitoxin.

The grand total gives 5,794 cases with 713 deaths, or a mortality of 12.3 per cent., including every case returned; but the reports show that 218 cases were moribund at the time of injection or died within twenty-four hours of the first injection. Should these be excluded there would remain 5,576 cases (in which the serum may be said to have had a chance) with a mortality of 8.8 per cent.

Of the 4,120 cases injected during the first three days there were 303 deaths—a mortality of 7.3 per cent., including every case returned. If from these we deduct the cases which were moribund at the time of injection, or which died within twenty-four hours, we have 4,013 cases, with a mortality of 4.8 per cent. Behring's original claim, that if cases were injected on the first or second day the mortality would not be 5 per cent., is more than substantiated by these figures. The good results obtained in third-day injections were a great surprise to your Committee. But after three days have passed the mortality rises rapidly, and does not differ materially from ordinary diphtheria statistics. Our figures emphasize the statement so often made, that relatively little benefit is seen from antitoxin after three days; however, it must be said that striking improvement has in some cases been seen even when the serum has been injected as late as the fifth or sixth day. The duration of the disease, therefore, is no contra-indication to its use.

TABLE I.—DAY OF INJECTION AND RESULT.

	Injected on 1st Day.			Injected on 2nd Day.			Injected on 3rd Day.			Injected on 4th Day.			Injected on or after 5th Day.			Day of Injection Unknown.			Totals.		
	Cases.	Deaths.	Mortality Per cent.	Cases.	Deaths.	Mortality Per cent.	Cases.	Deaths.	Mortality Per cent.	Cases.	Deaths.	Mortality Per cent.	Cases.	Deaths.	Mortality Per cent.	Cases.	Deaths.	Mortality Per cent.	Cases.	Deaths.	Mortality Per cent.
The Committee's Report.	764	38	4.9	1065	89	8.3	620	79	12 7	336	77	22.9	390	152	38.0	215	15	7.0	3384	450	13.0
New York Health Board.	126	11	8.7	215	26	12.0	228	37	16.6	153	32	20.9	203	59	20.0	17	4	23.5	942	169	17.8
Chicago Health Board.	106	0	0	336	5	1.5	660	18	2.7	269	38	14.1	97	33	34.0	0	0	0	1468	94	6.4
Totals.	996	49	4.9	1616	120	7.4	1508	134	8.8	758	147	20.7	690	244	35.4	232	19	8.2	5794	713	12.3

THE INFLUENCE OF BACTERIOLOGICAL DIAGNOSIS UPON THE STATISTICS.

This is shown in Table II.

TABLE II.—DIAGNOSIS CONFIRMED BY BACTERIOLOGICAL EXAMINATION.

Committee's Reports,	2,453 cases;	302 deaths;	mortality,	12.3	per cent.
N. Y. Board of Health,	916 "	160 "	"	16.9	" "
Chicago " "	1,468 "	94 "	"	6.4	" "
Totals, - -	4,837 "	556 "	"	11.4	" "
(Excluding 145 cases which were moribund or which died in twenty-four hours), - - -			"	8.7	" "

DIAGNOSIS FROM CLINICAL EVIDENCE ONLY.

Committee's Reports,	931 cases;	148 deaths;	mortality,	15.7	per cent.
N. Y. Board of Health,	26 "	9 "	"	34.6	" "
Totals - -	957 "	157 "	"	16.3	" "
(Excluding 72 cases either moribund or dying in twenty-four hours, - - - -			"	9.6	" "

In the cases in which the diagnosis was not confirmed by a bacteriological examination the mortality is thus 5 per cent. higher than in the bacteriological cases. This difference is to be explained by two facts: first, as already stated, that we have excluded from our reports all tonsillar cases (and hence most of the very mild ones) not confirmed by bacteriological examinations; and secondly, by the fact that this group of cases comprises those treated in the country where physicians have hesitated to use antitoxin unless the type of the disease was a grave one, and where also a large proportion of the injections were made later than in the cities. However, should we leave out the moribund cases, the mortality is but 9.6 per cent., which differs but slightly from the cases confirmed by bacteriological diagnosis.

In our subsequent statistics we shall consider together all the cases bacteriologically confirmed and otherwise, as the statistics are not materially altered by this grouping.

THE RESULTS AS MODIFIED BY THE AGE OF THE PATIENTS.

Unfortunately the ages have not been furnished in the report of the Chicago cases, and we have therefore only the cases reported to the Committee and those from the New York Board of Health for analysis. In Table III. are shown the mortality of the different ages grouped separately.

The highest mortality is seen as in all reports to be in the cases under two years, but including all those returned, even those that were moribund when injected, the death rate was but

TABLE III.—AGE AND RESULT OF TREATMENT.

	0 to 2 Years.			2 to 5 Years.			5 to 10 Years.			10 to 15 Years.			15 to 20 Years.			20 Years and Over.		
	Cases.	Deaths.	Mortality Per cent.	Cases.	Deaths.	Mortality Per cent.	Cases.	Deaths.	Mortality Per cent	Cases.	Deaths.	Mortality Per cent.	Cases.	Deaths.	Mortality Per cent.	Cases.	Deaths.	Mortality Per cent.
Committee's Report.	631	137	21.7	1276	75	13.7	8 83	108	12.2	276	19	6.8	112	4	3.6	214	9	4.2
New York Health Board.	236	65	27.5	466	83	17.8	178	21	11.2	29	0	0	11	0	0	22	0	0
Totals.	867	202	23.3	1742	258	14.7	1061	129	12.1	305	19	6.2	123	4	3.2	236	9	3.8
Moribund.	43			59			59			9			0			4		
Mortality Excluding Moribund Cases.			19.2			13.3			8.7			3.3			3.2			2.1

23.3 per cent., (21.7 per cent. of the Committee's cases) while if we exclude cases moribund when injected or dying within the first twenty-four hours, it falls to 19.2 per cent.

After the second year there is noticed a steady decline in mortality up to adult life. In many of the reports previously published the statement has been made that no striking improvement in results was observed in adult cases treated by the serum. Our figures strongly contradict this opinion. Of 359 cases over fifteen years old, which were returned, there were but thirteen deaths. That the reader may judge for himself how far antitoxin is to be held responsible for the result, a brief summary of these thirteen cases is appended.

Case I.—Fifteen years old; injected on the fourth day; membrane covering tonsils and pharynx; profoundly septic, sinking rapidly when injected; died in two hours. "My only death in seventeen cases" (Jones, Gloucester, Mass.).

Case II.—Forty-four years old; injected on the fourth day; membrane on the tonsils and pharynx; in bad condition; died three hours after injection. The tonsils had been previously incised, the early diagnosis having been quinsy.

Case III.—Thirty-one years old; injected on the sixth day; membrane on the tonsils, nose, pharynx, and larynx; intubation; sepsis; in bad condition; lived eight hours after injection.

Case IV.—Thirty-five years old; injected on the fifth day; membrane on the pharynx and nose (?); in bad condition; septic; died in twelve hours.

Case V.—Sixty years old; in bad condition; had serious mitral regurgitation; injected on the fourth day; membrane covering tonsils, pharynx, and larynx; died from heart failure on following day.

Case VI.—Sixty years old; "kidney trouble for years;" injected on the third day; very extensive membrane, covering tonsils, pharynx and nose; profound sepsis; in bad condition; died suddenly on the day after injection.

Case VII.—Seventeen years old; in bad condition; convalescing from measles; enormous adenopathy; profound sepsis; exceedingly high temperature; membrane covering tonsils and nose; injected at the the end of forty-eight hours; three injections, temporary improvement after each one; duration of life not given.

Case VIII.—Fifteen years old; in bad condition; injected on the ninth day; membrane covering tonsils, nose, pharynx, and larynx; no operation; enormous infiltration of the tissues of the neck; nephritis; sepsis; lived four days and died of sepsis.

Case IX.—Twenty years old; injected on the third day; membrane upon the tonsils, nose, pharynx and larynx; "a stubborn patient who got up before he was allowed, and died suddenly after it."

Case X.—Twenty-five years old; injected on the fifth day; membrane covering both tonsils, entire pharynx, and completely occluding nose; nephritis and sepsis; throat cleared off entirely; died suddenly on the fourteenth day from cardiac paralysis.

Case XI.—Nineteen years old; injected on the fifth day; membrane upon the tonsils and pharynx; profound sepsis; duration of life unknown.

Case XII.—Twenty-two years old; injected on the fourth day; membrane on the tonsils and gums; sepsis; died on the sixth day.

Case XIII.—The well-known Brooklyn case, reported in 1895. Girl, sixteen years old, who died suddenly ten minutes after injection.

Such are the adult cases which antitoxin failed to cure. Four of them were moribund at the time of injection, no one of them living over twelve hours. Two, both sixty years old, were already crippled by previous organic disease, one of the heart, and the other of the kidneys. In the measles case there was undoubted evidence of streptococcus septicæmia. Only two of the cases were injected as early as the third day; three of them on the fifth day; and one on the ninth day. Omitting the four moribund cases the mortality of 355 adult cases treated with the serum is 2.5 per cent.

PARALYSIS.

Reliable data upon this point and those hereafter to be mentioned are to be had only from the 3,384 reports returned to the Committee. Of these paralytic sequelæ appeared in 328 cases, 9.7 per cent. Of the 2,934 cases which recovered, paralysis was present in 276, or 9.4 per cent. Of the 450 cases which died, paralysis was noted in fifty-two, or 11.4 per cent.

The variety of the paralysis and the date of injection is shown in the following table:

TABLE IV.—VARIETY OF PARALYSIS AND THE DAY OF INJECTION.

RECOVERY CASES.	CASES.	DAY OF INJECTION.					
		1st Day.	2d Day.	3d Day.	4th Day.	5th Day.	Unknown.
Paralysis mentioned (variety not specified),	132	8	32	32	19	16	23
Throat only (aphonia, nasal voice or regurgitation),	114	16	21	25	11	16	24
Extremities,	14	3	5	2	—	3	1
Ocular,	11	—	4	3	1	2	1
General (multiple neuritis),	4	—	1	2	1	—	—
Sterno-mastoid,	1	—	1	—	—	—	—
FATAL CASES.							
Paralysis mentioned (variety not specified),	9	—	3	2	1	2	1
*Cardiac, late after throat clear (in 4 of them throat also),	32	1	2	8	9	8	4
Throat only,	6	—	2	—	—	—	4
General late,	4	—	1	—	1	2	—
Muscles of Respiration,	1	—	1	—	—	—	—
Totals,	328	28	73	76	43	49	59

Observations of some of the individual cases are interesting, particularly those of cardiac paralysis. It is twice stated that the child had gotten up and walked out of the house, where it was found dead. Twice death occurred after sitting up suddenly; once, on jumping from one bed into another. One patient of twenty years got up contrary to orders and died soon afterward. Another patient was apparently well until he indulged in a large quantity of cake and candy, soon after which cardiac symptoms developed, and he died shortly. One case was that of a woman sixty years old, who had serious organic cardiac disease.

It is difficult from these statistics to state what protective power the serum may have over the nerve cells and fibres. Apparently this is not great unless the injections are made early in the disease, and even then in severe cases the amount of damage done to these tissues in twenty-four hours may be very great, even irreparable. Time is not the only element in estimating the effect of the diphtheria toxines.

Great discrepancy exists in the statements made regarding the frequency of paralytic sequelæ after diphtheria. In a series of 1,000 cases reported by Lennox Browne, paralytic sequelæ

* Cases of heart failure occurring at the height of the disease have not been included here; although they are mentioned among the cases of cardiac paralysis in the table of fatal cases.

were present in 14 per cent. In 2,448 cases by Sanné, paralysis was noted in 11 per cent. In the series of cases here reported, the difference is slightly in favor of the antitoxin treatment, but paralysis is certainly frequent enough to show how extremely susceptible the nervous elements are to the diphtheria toxines. One thing is quite striking from a study of these cases, and that is the proportion that have died from late cardiac paralysis. That very many of them would undoubtedly have succumbed earlier in the disease from suffocation (laryngeal cases) or diphtheritic toxæmia, had the serum not been employed, is beyond question. Although the serum is able to rescue even many such desperate cases, it cannot overcome the effects of the toxines upon the cells, which have occurred before it was injected.

SEPSIS.

Sepsis is stated to have been present in 362 of the 3,384 cases or 10.7 per cent. It was present in 145 or 33 per cent. of the fatal cases. Some explanation is necessary for a correct appreciation of these figures. The majority of the reporters, it is plain from their remarks, have not distinguished between diphtheritic toxæmia and streptococcus sepsis. The former is certainly meant in the great majority of the cases. There is a very small proportion in which there is evidence of streptococcus sepsis. The six cases complicating measles, and the five complicating scarlet fever, however, should possibly be included among this list.

NEPHRITIS.

The statements on this point are quite unsatisfactory. The reports state that nephritis was present 350 times, or in 10 per cent. of the cases. On the one hand it must be stated that the diagnosis of nephritis rests in many cases simply upon the presence of albumen in the urine; but, on the other hand, it is true that in a large number of the cases, more than half, no examination of the urine is recorded as having been made, so that it is impossible to state with anything like approximate accuracy, the frequency of nephritis in these cases. Of the 450 fatal cases, the presence of nephritis is mentioned without qualification or explanation in thirty-nine cases; these being usually put down also as septic, dying in the acute stage of the disease. There are fifteen fatal cases, however, in which the renal disease was stated as the cause of death. In no less than nine the nephritis occurred

late in the disease, usually during the second or third week. In these fifteen cases the evidence of severe nephritis was conclusive, such symptoms being present as dropsy, suppression of urine, with coma or convulsions.

BRONCHO-PNEUMONIA.

Broncho-pneumonia is stated to have been present in 193 of the 3,384 cases, or 5.9 per cent., a remarkably small proportion when compared with hospital statistics. Among the patients that recovered, broncho-pneumonia was noted 114 times or in 3.8 per cent.; among the fatal cases seventy-nine times, or in 17.5 per cent., but in only about one-half of these was the pneumonia the cause of death. Of these thirty-seven were laryngeal cases operated upon late, ten were septic cases, and the pulmonary disease was coincident with the height of the diphtheritic process. In seven pneumonia was independent of both the above conditions. occurring late in the disease in all but two.

LARYNGEAL CASES.

Of the 3,384 cases reported to the Committee, the larynx is stated to have been involved in 1,256 cases or 37.5 per cent. This proportion is somewhat higher than is usual, and is partly explained by the fact that several physicians have sent in the reports only of their laryngeal cases. These laryngeal cases occurred in the practice of 379 physicians.

In 691, or a little more than one-half the number, no operation was done, and in this group there were 128 deaths. In forty-eight of them laryngeal obstruction was responsible for the fatal issue, operation being refused by the parents, or no reason for its being neglected having been given. In the eighty remaining fatal cases the patients died of other complications, and not from the laryngeal disease.

In the 563 cases, therefore, or 16.9 per cent. of the whole number, there was clinical evidence that the larynx was involved, and yet recovery took place without operation. In many of these cases the symptoms of stenosis were severe, and yet disappeared after injection without intubation. No one feature of the cases of diphtheria treated by antitoxin has excited more surprise among the physicians who have reported them, than the prompt arrest, by the timely administration of the serum, of membrane which was rapidly spreading downwards below the

larynx. Such expressions abound in the reports as "wonderful," "marvelous," "prepared to do intubation, but at my next visit the patient was so much better it was unnecessary," "in all my experience with diphtheria have never seen anything like it before," "no unprejudiced mind could see such effects and not be convinced of the value of the serum," etc., etc.

In establishing the value of the serum, nothing has been so convincing as the ability of antitoxin, properly administered, to check the rapid spreading of membrane downward in the respiratory tract, as is attested by the observations of more than 350 physicians who have sent in reports.

Turning now to the operative cases we find the same remarkable effects of the antitoxin noticeable. Operations were done in 565 cases, or in 16.7 per cent. of the entire number reported. Intubation was performed 533 times with 138 deaths, or a mortality of 25.9 per cent. In the above are included nine cases in which a secondary tracheotomy was done, with seven deaths. In thirty-two tracheotomy only was done with twelve deaths, a mortality of 37.4 per cent. Of the 565 operative cases, sixty-six were either moribund at the time of operation, or died within twenty-four hours after injection. Should these be deducted, there remain 499 cases operated upon by intubation or tracheotomy, with 84 deaths, a mortality of 16.9 per cent.

Of the 2,819 cases not operated upon, there were 312 deaths, a mortality of 11.3 per cent. Deducting the moribund cases, or those dying within twenty-four hours after injection, the total mortality of all non-operative cases was 9.12 per cent.

Let us compare the results of intubation in cases in which the serum was used, with those obtained with this operation before the serum was introduced. Of 5,546 intubation cases in the practice of 242 physicians, collected by McNaughton and Maddren (1892) the mortality was 69.5 per cent. Since that time statistics have improved materially by the general use (in and about New York, at least) of calomel fumigations. With this addition, the best results published (those of Brown) showed in 279 cases a mortality of 51.6 per cent.

Let us put beside the cases of McNaughton and Maddren the 533 intubations with antitoxin, with 25.9 per cent. mortality. With Brown's personal cases let us compare those of the fourteen observers who have reported to the Committee ten or more intubation operations in cases injected with serum. These com-

prise 280 cases with sixty-five deaths, a mortality of 23.2 per cent. In both comparisons the mortality without the serum is more than twice as great as in the cases in which serum was used.

The reports of some individual observers concerning intubation with the serum are interesting:

Neff, New York: twenty-seven operations, with twenty-seven recoveries.

Rosenthal, Philadelphia: eighteen operations, with sixteen recoveries.

Booker, Baltimore: seventeen operations, with seventeen recoveries, including one aged ten months, and one seven and a half months.

Seward, New York: eight operations, with eight recoveries.

McNaughton, Brooklyn: "In my last seventy-two operations without serum, mortality 66.6 per cent.; in my first seventy-two operations with serum, mortality 33.3 per cent."

O'Dwyer, New York: "In my last 100 intubations, first seventy, without serum, mortality 73 per cent.; last thirty, with serum, mortality 33.3 per cent."

But even these figures do not adequately express the benefit of antitoxin in laryngeal cases. Witness the fact that over one-half the laryngeal cases did not require operation at all. Formerly 10 per cent of recoveries was the record for laryngeal cases not operated upon. Surely, if it does nothing else the serum saves at least double the number of cases of laryngeal diphtheria that has been saved by any other method of treatment.

The great preponderance of intubation over tracheotomy operations shows how much more highly the profession in this country esteems the former operation.

A STUDY OF THE FATAL CASES.

Of the 450 fatal cases in the Committee's Report, 229, or one-half, received their first injection of the serum on or after the fourth day of the disease, and 152, or over one-third of these, on or after the fifth day.

There were fifty-eight cases in which it was stated that the child was moribund at the time of injection, the serum being administered without the slightest expectation of benefit, but at the earnest solicitation of the parents.

There remain 350 cases in which the cause of death could be pretty accurately determined by the reports. These died from

the following causes, the most important cause being placed first:

Sepsis (*including diphtheritic toxæmia*) was the cause of death in 105 cases; of which sixteen had nephritis, four were intubated or tracheotomized, two were laryngeal cases not operated upon, four had paralysis, one had pneumonia, and in one the fatal sepsis was attributed to a traumatic condition of the left knee.

Cardiac paralysis was the cause of death in fifty-three cases. Under this head are included cases of sudden heart failure occurring at the height of the disease (twenty-one in number) as well as those more commonly designated as heart paralysis, where death occurred suddenly after the throat cleared off. Of the latter there were thirty-two examples; four of these cases had throat paralysis, nineteen were septic, eight had nephritis, five were intubated, and one tracheotomized.

Broncho-pneumonia was put down as the cause of death in fifty-four cases. In thirty-seven of these it followed laryngeal diphtheria; of these twenty-two were intubated, and four tracheotomized; two had nephritis; nine were septic. Broncho-pneumonia and sepsis was the cause of death in ten cases, of which three had nephritis and one general paralysis. Broncho-pneumonia caused death in seven cases, apart from sepsis or laryngeal diphtheria; of these only one had nephritis; one died from heart failure; and in five pneumonia came on late in the disease.

Laryngeal diphtheria without operation caused death in forty-eight cases. In some of these the operation was refused by the parents, in others it was neglected by the physician, the patients dying of asphyxia; three of these cases had nephritis, four were septic, two had pneumonia, and one had sepsis and nephritis.

Diphtheritic tracheitis or bronchitis caused death in eleven cases; all of these were intubated, and in two there was evidence of the existence of membrane in the bronchi before operation. There were thirty-three other cases in which death followed laryngeal diphtheria without the supervention of pneumonia. It is highly probable that in some of these death was due to membranous tracheitis or bronchitis. All of them were operated upon; ten were septic, two had paralysis, and one had nephritis.

Sudden obstruction of the intubation tube was the cause of death in three other laryngeal cases.

The tube was coughed up in three cases, fatal asphyxia occurring before the physician could be summoned.

Died on the table during tracheotomy, one case.

Nephritis was the cause of death in fifteen cases; seven of these were septic, and three had been intubated.

General paralysis was the cause of death in five cases; in all probably the pneumogastric was involved.

Paralysis of the respiratory muscles produced death in one case, one of laryngeal diphtheria, which was intubated, and was complicated by broncho-pneumonia.

Measles associated with diphtheria produced death in six cases; five of these were laryngeal and were intubated; in two there was pneumonia, and in two sepsis. Diphtheria developed during the height of the measles, or immediately followed it.

Scarlet fever with diphtheria was the cause of death in six cases; in three of these there was broncho-pneumonia, nephritis and sepsis; in two scarlet fever preceded diphtheria, and in one of these there was sepsis with gangrene of the tonsils. In the sixth case the patient died of scarlet fever, which developed during convalescence from the diphtheria.

Gangrene of the cervical glands or cellular tissue of the neck was the cause of death in two cases associated with profound general sepsis.

Endocarditis caused death in one case, nineteen days after the diphtheria.

Diphtheritic inflammation of the tracheal wound with sepsis caused death in one case.

General tuberculosis, five weeks after diphtheria, was assigned as the cause of death in one case.

Exhaustion was the cause of death in three cases, one a protracted case; another complicated by pneumonia and sepsis; one by nephritis.

Convulsions was the cause of death in three cases apart from disease of the kidneys. In one, the well-known Brooklyn case, the girl died ten minutes after the injection, in another twenty-four hours after injection, in the third the particulars were not given.

Meningitis was assigned as the cause of death in one case.

THE KIND OF ANTITOXIN USED.

They are given in the order of frequency with which they have been used. First, the serum prepared by the New York Board of Health; second, Behring's; third, Gibier's;* fourth, Mulford's; fifth, Aronson's; sixth, Roux's. In addition a large number of cases are reported as having been treated by the serum prepared by the Health Boards of different cities—Brooklyn, Newark, Rochester, Pittsburgh, etc. The largest number of cases have been treated by the serum prepared by the New York Health Board, a very large number by Behring's serum, all others being relatively in small numbers.

Dosage and number of injections. In the great majority of cases but one injection is reported. In very severe ones two and three have been given. The largest amount is in a case by Weimer (Chicago) who gave eighteen injections of Behring's serum to a laryngeal case in a child thirteen years old. Another instance of ten injections is reported with no unfavorable symptoms.

As a rule the dosage has been smaller in antitoxin units than is now considered advisable, particularly in many of the laryngeal cases and others injected later than the second day.

CASES INJECTED REASONABLY EARLY (DURING THE FIRST THREE DAYS) IN WHICH ANTITOXIN IS SAID TO HAVE PRODUCED NO EFFECT, THE DISEASE ENDING FATALLY.

These cases are nineteen in number. Brief reports are introduced that the reader may judge to what degree they may be regarded as a test of the serum treatment. In our statistical tables all of them have been included among the fatal cases.

In Cases I. and II. the cultures were reported negative. Case I., by Gallagher, New York: Child, eighteen months old; septic; although no eruption was present, the reporter was "inclined on reflection to regard this case as one of scarlatinal sore throat."

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*It is worthy of note that in the tests made by the State Board of Health of Massachusetts, published under date of April 6, 1896, this serum was found far below the standard as labelled upon the bottle; thus a package marked to contain 2,500 units, by test was found to contain less than 700. All the other varieties of serum tested were found essentially up to the standard.

Case II., by Potter, Buffalo: Male, fourteen months old; two cultures made, but no Löffler bacilli found; membrane in the nose and pharynx. Injected on the third day, one dose of Behring's serum No. 1. No improvement; death from sepsis. "Probably pseudo-diphtheria" (I. H. P.).

In Cases III. to IX. no cultures were made.

Case III., by Tefft, New Rochelle: Seven years old; injected after eighteen hours' illness; two injections of Behring's No. 2 serum; membrane on the tonsils, pharynx, and nose; no effect observed from injections; patient dying on the third day.

Case IV., by Tefft: Male, four years old; membrane on the tonsils and pharynx; injected after thirty-six hours' illness with Behring's No. 2; died on the third day; no noticeable effect from the injection.

Case V., by Tefft: Six years old; membrane on the tonsils, nose, and pharynx; septic; injected after thirty-six hours' illness; three injections of Behring's No. 2. "Saw no effect from the injections, the disease going steadily on to a fatal termination."

Case VI., by Cameron, Montreal: Two and a half years old; fifty hours ill; membrane on the tonsils, nose and pharynx; septic; no improvement noticed, and child died twenty hours after injection.

Case VII., by Baker, Newtonville, Mass.: Three years old; laryngeal diphtheria; injected on the third day 10 c.c. Roux's serum; cyanosis; intubation; temperature 103° F., and continued high until death in eighteen hours after operation; injections had no effect.

Case VIII., by Anderson, New York: Three years old; injected after three hours' illness; membrane on the tonsils, nose and pharynx; one injection New York Health Board antitoxin. "A case of malignant diphtheria, full duration twenty-four hours."

Case IX., by McLain, Washington: Four years old; twelve hours sick; membrane on the pharynx and larynx; two injections; no operation; first injection early in the morning, the other early in the afternoon; died the same day; no change in the condition; antitoxin had no apparent effect.

In Cases X. to XIII. diphtheria complicated measles, all reported by W. T. Alexander, New York. Disease confined to the larynx in all; in three the stenosis developed during measles, and in one while the patient was convalescing from measles;

diagnosis confirmed by culture in every case, and in all intubation performed. Antitoxin seemed to have no effect, the cases going on to a fatal termination; all received their injections within twenty-four hours after the laryngeal symptoms appeared.

In three cases—XIV. to XVI.—the type of the disease was malignant from the outset.

Case XIV., by Lloyd, Philadelphia: Fifteen months old; injected after thirty-six hours' illness; diagnosis confirmed by culture; membrane covered the tonsils, pharynx, nose, and larynx; intubation; sepsis; death on the fifth day. Although antitoxin was used as promptly as possible no perceptible effect noticed. One injection, Behring's No. 3, was given.

Case XV., by Wert, Mount Vernon, N. Y.: Eighteen months old; injected on the third day; diagnosis confirmed by culture; membrane on the tonsils and pharynx. "Very intense type of the disease." Antitoxin could not be procured before the third day; Gibier's serum used. "Died suddenly in apparent convulsions about ten hours after injection; urine not examined; very little passed."

Case XVI., by Ingraham: Six years old; membrane covered the tonsils, pharynx, and larynx; diagnosis confirmed by culture; pneumonia present; condition very bad; injected after two and a half days' illness; three injections of Behring's serum; no benefit noticed.

Case XVII., by Johnson, Buffalo: Three years old; twelve hours ill; case septic from the start; membrane on the tonsils, pharynx, and larynx; diagnosis confirmed by culture. "Antitoxin apparently had very little effect."

Case XVIII., by Baker, Newtonville, Mass.: Two and a half years old; twenty hours ill; disease confined to larynx; diagnosis confirmed by culture; one injection of Gibier's serum; intubation. "Was doing well a few minutes before death when child got up in its crib, changed color and died almost immediately." Death attributed to "sudden heart failure; found no obstruction of the tube."

Case XIX., by Story, Washington: Five years old; in fair condition; thirty-six hours ill; diagnosis confirmed by culture; membrane on the tonsils, pharynx, and larynx; one injection of United States Marine Hospital antitoxin; injection produced no effect.

CASES IN WHICH UNFAVORABLE SYMPTOMS WERE, MIGHT HAVE BEEN, OR WERE BELIEVED TO HAVE BEEN, DUE TO ANTITOXIN INJECTIONS.

Only three cases reported to the Committee could by any possibility be placed in this category. All of the details furnished by the reporters are reproduced:

Case I., by Kortright, Brooklyn: Sudden death in convulsions ten minutes after injection. This case is the already well-known Valentine case, occurring in Brooklyn in the spring of 1895. The principal points were as follows: A girl sixteen years old; in good condition; tonsillar diphtheria; diagnosis confirmed by culture; injected on the first day with 10 c.c. Behring's serum; died in convulsions ten minutes later.

Case II., by Kerley, New York: Fairly healthy boy, two and one half years old; membrane on tonsils, pharynx and in nose. Diagnosis confirmed by culture; injected on the morning of the fourth day with 10 c.c., (1000 units) New York Health Board serum; temperature at time of injection 100.4° F; no sepsis, and child apparently not very sick; urine free from albumen. Distinctly worse after injection; in ten hours temperature rose to 103° F.; urine albuminous; throat cleared off rapidly, but marked prostration and great anæmia, with irregular fluctuating temperature continued and death from exhaustion with heart failure four days after the use of the serum.

Case III., by Eynon, New York: Male, three and one half years old; diagnosis confirmed by culture; two days ill; membrane on tonsils and in nose; two injections New York Health Board serum. "A rapid nephritis developed after the second injection causing coma, convulsions and death twenty hours after the second injection." In response to an inquiry for further particulars the following was received: "The case seemed a mild one, but the injection was given one afternoon and repeated the following afternoon, about 1500 units in all. The urine up to that time had not been examined. About fourteen or sixteen hours after the second injection unfavorable symptoms began to develop pointing to infection of the kidneys. The urine was found to be loaded with albumen. My impression at the time was that the antitoxin either produced, hastened or intensified nephritis, thereby causing the fatal termination."

In regard to the three fatal cases just cited, Case I., is wholly unexplained. In Case II., the query arises, did this sudden change hinge upon the injection of the serum, or was it one of those unexplained abrupt changes for the worse in a case apparently progressing favorably, so often observed in diphtheria? As regards Case III., it will be seen from the letter that the evi-

dence is not at all conclusive. All details available are given, and the reader may draw his own conclusions.

CLINICAL COMMENTS.

The following are selected from hundreds which have been received, and may be taken fairly to represent the sentiments of the physicians who have sent in reports:

Dr. Douglas H. Stewart, New York, sends reports of four cases, all desperate ones, and all "presumably fatal under any other form of treatment." Very extensive membrane in all; larynx involved in three; in one neglected case in a child three years old, *injected upon the fifth day*, the membrane covered the tonsils, nose, pharynx, and larynx. Broncho-pneumonia, nephritis and sepsis all present. Temperature 107°F. at the time of the first injection. Prostration so great that he dared not attempt intubation. Believes that this case would certainly have been fatal in a few hours without antitoxin. Perfect recovery.

In another case three years old, membrane first discovered in the left ear, next morning seen upon the tonsils. and spread in a few hours over the pharynx into the larynx and trachea. Intubation necessary in a few hours; had never seen membrane spread so rapidly as in this child. Urine albuminous; membrane subsequently expelled from larynx and trachea in large casts, with profuse bloody expectoration. Complete recovery on the ninth day. The physician describes this as "the very worst case of diphtheria that has ever come under my notice." Five thousand four hundred antitoxin units were given in four injections. He remarks: "My experiences in the past have been so very unfortunate that the advocates of antiseptics or therapeusis were a constant surprise to me. It has been my fate to have the most desperate cases unloaded upon my shoulders. I had been forced into the belief that the profession was absolutely powerless in the presence of true diphtheria; have lost case after case with tube in the larynx and calomel fumigations at work. Previous to antitoxin my only hope had become centered in nature and stimulants. In two years have not lost a single case, and surely I may be pardoned if I suffer from diphtheria-phobia in a sub-acute form, and use antitoxin sometimes unnecessarily."

Dr. L. L. Danforth, New York, states that during his twenty-two years of practice in New York he has seen many fatal cases of diphtheria, had used all kinds of remedies, mainly those of the homeopathic school, and while he had as much confidence in the latter as in anything else, he had seen so many deaths during the year past that he "hailed with delight the advent of antitoxin, and determined to use it." Reports five cases, all of a severe type. "The result in every case has been marvelous. I would not dare to treat a case now without antitoxin."

Dr. H. W. Berg, New York, reporting fourteen cases, says: "I have not yet ceased to be surprised at the recovery of some of these cases, which, in the light of my former experience with diphtheria treated without antitoxin, seemed to be irretrievably lost."

Dr. George McNaughton, Brooklyn, reports seventy-two laryngeal cases, with twenty-four deaths; sixty-seven of these were intubated, with twenty-one deaths. He states that he has kept no records of cases other than laryngeal ones, as these seemed the best test of the serum treatment. He believes that if the serum is used early, very many cases will not need operation for the relief of stenosis. "I would urge the use of antitoxin in all cases of croup in any patient who has an exudation upon the pharynx; would not wait for bacteriological confirmation of diagnosis, for in so doing valuable time is lost." Has noticed that the tube is coughed up more frequently in injected cases, and believed this due to the fact that the swelling of the tissues subsides at an earlier date.

Dr. D. C. Moriarta, Saratoga, reporting four cases, says that the first was a malignant one and "I only used the remedy because I am Health Officer and was urged to do so, as the type of the disease was that from which I have seen recovery but once in eleven years." Boy five years old, four days ill when injected; great prostration, rapid breathing, and he was "practically gone." Nares filled and tonsils and pharynx covered; severe nasal hemorrhage; cervical glands greatly swollen; heart's action very frequent and feeble; child unable to lie down. Behring's serum twenty c.c. injected; in six hours evidently more comfortable; in eighteen hours decidedly improved; in twenty-four hours sitting up and feeling much better; in forty-eight hours all urgent symptoms gone and membrane loosening. Subsequently had nephritis which lasted six weeks, and multiple neuritis which persisted for three months, but ultimately recovered perfectly. "I send this report because it converted me. No unbiased person familiar with diphtheria could see such results as this and not feel there must be good in it."

Dr. F. M. Crandall, New York, sends report of a child seven years old. Membrane on the tonsils and in larynx, with croup for forty hours when antitoxin was injected and intubation done. Progress of the disease had been rapid; semi-stupor and eyes half open; very feeble rapid pulse; intense toxæmia; general cyanosis. Both cyanosis and dyspnœa persisted after intubation, showing clearly the presence of membrane below the tube. Case regarded as "absolutely hopeless." The first change was seen in the disappearance of toxæmia, with improvement in the pulse, clearness of the mind, etc.; later a change in the local condition; large masses of membrane were expelled from the larynx and trachea, necessitating frequent removals of

the tube. Tube finally removed in a week with complete recovery.

Dr. Reynolds, Baltimore, mentions a case showing the danger of relying too implicitly upon the bacteriological diagnosis. Male, three years. Culture reported only staphylococcus and streptococcus, consequently injection delayed until the fifth day, when membrane covered tonsils, nose, and pharynx. Child died two days later. A sister subsequently contracted the disease, received antitoxin on the third day and recovered. The reporter would not wholly rely upon the culture test for diagnosis.

SUMMARY.

(1) The report includes returns from 615 physicians. Of this number more than 600 have pronounced themselves asstrongly in favor of the serum treatment, the great majority being enthusiastic in its advocacy.

(2) The cases included have been drawn from localities widely separated from each other, so that any peculiarity of local conditions to which might be ascribed the favorable reports must be excluded.

(3) The report includes the record of every case returned except those in which the evidence of diphtheria was clearly questionable. It will be noted that doubtful cases which recovered have been excluded, while doubtful cases which were fatal have been included.

(4) No new cases of sudden death immediately after injection have been returned.

(5) The number of cases injected reasonably early in which the serum appeared not to influence the progress of the disease was but nineteen, these being made up of nine cases of somewhat doubtful diagnosis; four cases of diphtheria complicating measles, and three malignant cases in which the progress was so rapid that the cases had passed beyond any reasonable prospect of recovery before the serum was used. In two of these the serum was of uncertain strength and of doubtful value.

(6) The number of cases in which the patients appeared to have been made worse by serum were three, and among these there is only one new case in which the result may fairly be attributed to the injection.

(7) The general mortality in the 5,794 cases reported was 12.3 per cent.; excluding the cases moribund at the time of injection or dying within twenty-four hours, it was 8.8 per cent.

(8) The most striking improvement was seen in the cases injected during the first three days. Of 4,120 such cases the mortality was 7.3 per cent.; excluding cases moribund at the time of injection or dying within twenty-four hours, it was 4.8 per cent.

(9) The mortality of 1,448 cases injected on or after the fourth day was 27 per cent.

(10) The most convincing argument, and, to the minds of the Committee, an absolutely unanswerable one in favor of serum therapy, is found in the results obtained in the 1,256 laryngeal cases (membranous croup). In one-half of these recovery took place without operation, in a large proportion of which the symptoms of stenosis were severe. Of the 533 cases in which intubation was performed the mortality was 25.9 per cent., or less than half as great as has ever been reported by any other method of treatment.

(11) The proportion of cases of broncho-pneumonia—5.9 per cent.—is very small and in striking contrast to results published from hospital sources.

(12) As against the two or three instances in which the serum is believed to have acted unfavorably upon the heart, might be cited a large number in which there was a distinct improvement in the heart's action after the serum was injected.

(13) There is very little, if any, evidence to show that nephritis was caused in any case by the injection of serum. The number of cases of genuine nephritis is remarkably small, the deaths from that source numbering but fifteen.

(14) The effect of the serum on the nervous system is less marked than upon any other part of the body; paralytic sequelæ being recorded in 9.7 per cent. of the cases, the reports going to show that the protection afforded by the serum is not great unless injections are made very early.

The Committee feels that this has been such a responsible task that it has thought best to state the principle which has guided it in making up the returns. While it has endeavored to present the favorable results with judicial fairness, it has also tried to give equal or even greater prominence to cases unfavorable to antitoxin.

In conclusion the Committee desires in behalf of the Society to express its thanks to members of the profession who have

cöoperated so actively in this investigation, and to Dr. A. R. Guerard for the preparation of the statistical tables.

(Signed)

L. Emmett Holt, M.D.,
W. P. Northrup, M.D.,
Joseph O'Dwyer, M.D.,
Samuel S. Adams, M.D.,
} Committee.

THE ACTION OF THE SOCIETY UPON THE REPORT.

At the close of its presentation, the Society voted to accept the report of the Committee and after a full discussion it was decided to embody its conclusions in the following resolutions:

(1) *Dosage.* For a child over two years old, the dosage of antitoxin should be in all laryngeal cases with stenosis, and in all other severe cases, 1500 to 2000 units for the first injection, to be repeated in fron eighteen to twenty-four hours if there is no improvement; a third dose after a similar interval if necessary. For severe cases in children under two years, and for mild cases over that age the initial dose should be 1000 units, to be repeated as above if necessary; a second dose is not usually required. The dosage should always be estimated in antitoxin units and not of the amount of serum.

(2) *Quality of Antitoxin.* The most concentrated strength of an absolutely reliable preparation.

(3) *Time of administration.* Antitoxin should be administered as early as possible on a clinical diagnosis, not waiting for a bacteriological culture. However late the first observation is made, an injection should be given unless the progress of the case is favorable and satisfactory.

The Committee was appointed to continue its work for another year and was requested to issue another circular asking for the further cöoperation of the profession, this circular to be sent out as soon as possible in order that physicians may record their cases as they occur through the coming year.

COMPARATIVE RESULTS OF THE TREATMENT OF DIPHTHERIA, WITH AND WITHOUT ITS ANTITOXIN, IN THE DISTRICT OF COLUMBIA.*

BY SAMUEL S. ADAMS, M.D.

Professor of Pediatrics, University of Georgetown; Visiting Physician, Children's Hospital, Washington, D. C.

In the prosecution of the work of collecting the data relative to the treatment of diphtheria, with its antitoxin, in private practice in the District of Columbia, cases of diphtheria in which the serum was not used formed such a large proportion of the whole number that they have been incorporated in this paper for purposes of comparison. It must be understood that information asked for in the circular was not sought in the latter class of cases. All cases—321, of diphtheria except ten—officially recorded, as occurring in the District of Columbia from January 1, 1895, to April 15, 1896, are included in this paper. The exclusion of seven of the exceptional cases was because bacilli diphtheriæ were present without the membrane, and the injection was used for immunization; and in the other three it was impossible to obtain any information from the two reporters which would enable classification. While the physicians themselves have furnished the details in the cases treated with serum, the official records of the Health Department have been relied upon in the other cases.

During the period named eighty-three physicians treated 176 cases of diphtheria with antitoxin. Of these seventy-one reported 160 cases in full, while twelve, who had treated the remaining sixteen cases, did not respond. The latter, however, are covered in this report, as sufficient information was gleaned from other sources to warrant proper classification. During the same time eighty physicians treated 135 cases of diphtheria without using its antitoxin. A third class of twenty-two physicians used the antitoxin in fifty-six cases, but did not use it in thirty-eight.

Ages.—As the investigation was confined to children the sixteenth year represents the maximum age. The youngest child receiving the antitoxin was aged ten months, and the eldest sixteen years, the average age being 6.51 years.

The ages of these children is shown by the following table: 6 months to 1 year. 1; 2 years, 6; 3 years, 15; 4 years, 10: 5

* Read before the American Pediatric Society, Montreal, May 26, 1896.

years, 21; 6 years, 17; 7 years, 16; 8 years, 20; 9 years, 18; 10 years, 20; 11 years, 9; 12 years, 4; 13 years, 3; 14 years, 3; 15 years, 3; 16 years, 4; total 176.

The youngest child treated without antitoxin was four months old, and the eldest fifteen years, the average age being 5.96 years.

Table showing ages of those not receiving antitoxin: Under 6 months, 1; 1 year, 1; 2 years, 7; 3 years, 12; 4 years, 23; 5 years, 14; 6 years, 14; 7 years, 12; 8 years, 10; 9 years, 6; 10 years, 10; 11 years, 10; 12 years, 3; 13 years, 6; 14 years, 4; 15 years, 2; total 135.

Sex and Color are shown by the following table:

	Treated with Antitoxin.	Treated without Antitoxin.
White Male, ...	78	52
" Female	85	56
Colored Male	3	11
" Female	10	16
Total	176	135

While girls seem to be more susceptible to diphtheria in the proportion of 167 to 144 boys, the infrequency of the disease in the negro is most astonishing. The negroes affected represent but 12.54 per cent. of the whole number.

Physical Condition.—The following table is supposed to show the physical condition of the patient at the time the antitoxin was injected. Although the exact condition given by the reporter was recorded in every case, it cannot be accepted as being correct, owing to discrepancies in other statements. For instance, some reported "good," and further on "sepsis" and "death" within twenty-four hours; others as "fair," when the comments showed the patient cyanozed, requiring immediate intubation; and still others "bad," although the injection was given a "few hours" after the appearance of the membrane. Thus the purport of the question referring to the physical condition of the patient at the time of injecting the antitoxin was undoubtedly misunderstood by many.

Good.	Fair.	Bad.	Not Stated.	Total.
73	41	52	10	176

Time of Injecting Antitoxin.—The following table shows the time elapsing between the first appearance of the membrane and the injection of the antitoxin. A vast majority answered this question correctly, but a few understood it to apply to the time

between the diagnosis and the injection. The importance of being accurate in this observation will be appreciated in studying the results of this treatment.

6 hrs.	12 hrs.	24 hrs.	36 hrs.	2 d.	3 d.	4 d.	5 d.	6 d.
2	3	26	12	31	29	21	9	8

7 d.	8 d.	9 d.	10 d.	11 d.	Not stated.	
11	1	1	1	1	20	Total 176.

Thus 103 cases received the injection on or before the third day of the disease.

Diagnosis.—The diagnosis was confirmed by a bacteriological examination in 159 cases and was made without it in seventeen.

Number of Injections given in each case.—One injection was administered in each of 119 cases; two in thirty-four cases; three in five cases; seven in one case; and the number of injections was not stated in seventeen cases.

Operations.—Intubation was performed twelve times and tracheotomy once; the latter having been performed after the expulsion of the intubation-tube which had been in place seven hours.

Brief notes of these operations may prove of interest. The reporter and operator are not mentioned to avoid making invidious distinctions since numerous cases quite as interesting and not requiring an operation, have also been reported:

Case I.—A. G., white, female, aged two years, was in bad condition on the fourth day, when two injections of Behring's serum were given. The membrane extended over tonsils, pharynx and larynx, necessitating intubation. The diagnosis was confirmed by cultures. Broncho-pneumonia and sepsis complicated the case at the time of injection. The serum "acted well," slight paralysis supervened and recovery was complete.

Case II.—F. H., white, male, aged three years, was in dying condition when the serum was administered. The membrane involved the larynx and dyspnœa was so severe that intubation was performed at the time of injection. The urgent symptoms were relieved, but the child died from strangulation in attempting to feed him a few hours after the introduction of the tube.

Case III.—R. G., white, female, aged three years, nine months, was in very bad condition on the middle of the eighth day when the reporter took charge of the case. The membrane involved the tonsils and larynx and two injections of Behring's serum were given. The diagnosis was confirmed by culture, the laryngeal symptoms grew worse, and intubation was performed on the tenth day of the disease. The child recovered.

Case IV.—X. J., white, male. aged four years, was in very bad condition on the fifth day, sepsis being noted. The membrane involved the tonsils, nose, pharnyx and larynx. The diagnosis was not confirmed by culture, but one injection of Behring's serum was administered. The laryngeal obstruction required intubation, but the tube was expelled in seven hours, when tracheotomy was performed. The child died.

Case V.—M. L., white, male, aged four years, was septic and in a moribund state when first seen by the reporter. The membrane involved the pharynx and larynx. The diagnosis was confirmed by culture. One injection of Behring's serum was administered; six hours later intubation was performed, but the patient died twenty hours after the injection.

Case VI.—C. K., white, female, aged four and one-half years, was in good condition when first seen. The membrane involved the tonsils, nose, pharynx and larynx. The diagnosis was confirmed by culture. Two injections of Behring's serum were administered and intubation performed. Nephritis complicated matters, but recovery was complete.

Case VII.—V. C., white, male, aged four years, was in bad condition on the third day after the recognition of the membrane when two injections of antitoxin were administered, which "acted well." The membrane was located in the larynx, making intubation necessary. The diagnosis was confirmed by culture. Sepsis was present, but the child recovered. This patient had measles just after his recovery from diphtheria.

Case VIII.—L. B., white, female, aged six years, was in bad condition on the third day, when the first injection of Behring's serum was given. The following morning she received the second injection. The membrane involved the tonsils and larynx. The diagnosis was confirmed by culture. At noon of the fourth day the patient was cyanozed, semi-conscious and death from suffocation seemed imminent. Intubation relieved the dyspnœa, and recovery was speedy.

Case IX.—W. D., white, male, aged six years, was in a fair condition on the fourth day when one injection of Behring's serum was administered. The membrane involved the tonsils and larynx and was undoubtedly diphtheritci, although the diagnosis was not confirmed by culture. Intubation was performed, and the child recovered.

Case X.—W. F., white, male, aged seven years, was in good condition on the fifth day when two injections of Behring's serum were administered. The membrane involved the tonsils, pharynx and larynx. The diagnosis was confirmed by culture. Intubation was performed and the child recovered.

Case XI.—M. M., white, male, aged seven years, was in a very bad state on the second day when one injection of Behring's serum was administered. The membrane involved the tonsils, nose, pharynx and larynx. The diagnosis was confirmed by

culture. Intubation was performed but the tube was expelled in forty-eight hours. The patient recovered without the re-introduction of the tube, although paralysis supervened.

Case XII.—H. T., white, female, aged eight years, was in very bad condition thirty-six hours after the appearance of the membrane, when three injections of Behring's serum were administered. The membrane involved the tonsils, pharynx and larynx. The diagnosis was confirmed by culture. Intubation was performed and the patient recovered.

The three fatal cases in which intubation was performed require some comment. In one, aged three years, the alarming symptoms were relieved, but the patient, unfortunately, died from strangulation before any idea of the action of the antitoxin could be formed. In another, aged four years, the injection was administered, and intubation and tracheotomy were performed on the fifth day after the recognition of the disease. The physician pronounced this case hopeless upon his first visit, but must be commended for his efforts to relieve the child's suffering by the second operation. In the third, aged four years, the child's condition and surroundings were such that death was imminent when the physician first saw him, but nevertheless, intubation was performed and the antitoxin administered.

The other cases of diphtheria, which recovered after intubation, were unusually severe; seventy-five per cent. recoveries of intubation in children between two and seven years is more than creditable and must be greatly due to the beneficial effects of the antitoxin.

Location and extent of the membrane.—The location of the membrane has been accurately recorded as follows:

Tonsils, 46; larynx, 3; tonsils and pharynx, 34; nose, pharynx and larynx, 2; tonsils and nose, 8; pharynx, 3; tonsils and larynx, 11; pharynx and larynx, 3; nose and larynx, 1; nose and pharynx, 2; tonsils, pharynx and larynx, 11; tonsils, nose and larynx, 1; tonsils, nose, pharynx and larynx, 15; tonsils, nose and pharynx, 19; nose, 1; not stated, 16; total, 176.

Complications.—The following are the complications in the order of their frequency: Nephritis in 13; paralysis in 13; sepsis in 12; nephritis and paralysis in 5; broncho-pneumonia in 3; sepsis and paralysis in 3; nephritis and sepsis in 2; nephritis, sepsis and paralysis in 2; broncho-pneumonia, sepsis and paralysis in 1.

Only three cases of erythema are reported. Scarlatinal infection complicated two cases, one being fatal.

Results.—That which concerns us most in a discussion of this kind is the therapeutic value of the antitoxin of diphtheria. One reporter may assert that it acts as if by magic; another that he could see nothing astonishing in its action, but has no objection to using it; while a third, the defender of the time honored compound of the ferric chloride and potassium chlorate, decries its use altogether. The members of each class are entreated to ponder over the following data before passing final judgment.

During the year ending June 30, 1894, before the introduction of antitoxin, 432 cases of diphtheria were reported in this district, the average age of those attacked being 8.7 years. Fifty-eight were white males, fifty-four white females, twenty-six colored males and thirty-four colored females. There were 172 deaths, the percentage of deaths to cases being 39.8.

During the fifteen and a half months ending April 15, 1896, after the introduction of antitoxin, 311 cases of diphtheria were reported, the average age of those attacked being 6.23 years. There were seventy-three deaths, the percentage of deaths to all cases being 23.44.

During the same period 135 cases were treated without antitoxin, the average age being 5.96 years. There were forty-seven deaths, the percentage of deaths to all such cases being 34.81—a death-rate of only 4.99 per cent. less than that when antitoxin was unknown—the average age of the decedents being 4.25 years.

During this period also, 176 cases were treated by the injection of antitoxin, the average age being 6.51 years. There were twenty-six deaths, the percentage of deaths to all cases receiving the antitoxin being 14.77, the average age of the decedents being 4.66 years.

There were twenty-two physicians who used antitoxin, in fifty-six cases, but did not use it in thirty-eight cases during the same period. In the former group there were fourteen deaths, the percentage of deaths to such cases being twenty-five; in the latter group there were eleven deaths, the death rate being 28.94 per cent.

The reporters' notes on the fatal cases in which antitoxin was used may be summarized as follows: 2 years, 2; 2½ years, 2; 3 years, 4; 3½ years, 2; 4 years, 6; 5 years, 2; 6 years, 3; 7

years, 2; 8 years 1; 9 years, 1. The youngest child was two years old, and the eldest nine years, the average age being 4.66 years.

The ages of the decedents that did not receive antitoxin are given below: 5 months, 1; 9 months, 1; 1 year, 3 months, 2; 1 year, 5 months, 1; 1 year, 6 months, 2; 1 year, 8 months, 1; 2 years, 5; 2 years, 6 months, 2; 2 years, 9 months, 1; 3 years, 5; 3 years, 6 months, 1; 3 years, 10 months, 1; 4 years, 4; 4 years, 11 months, 1; 5 years, 3; 5 years, 2 months, 1; 6 years, 4; 6 years, 3 months, 1; 7 years, 2; 7 years, 3 months, 1; 8 years, 1; 9 years, 3; 10 years, 1; 10 years. 3 months, 1; 12 years, 1. The youngest was six months old and the eldest twelve years, the average age being 4.25 years.

Fifteen white males, nine white females, and two colored females, that had received antitoxin died. While of those treated without antitoxin, twenty white males, sixteen white females, five colored males and six colored females died. The physical condition at the time of the injection was stated to be good in three, fair in two, bad in nineteen and was not given in two. The time from the first appearance of the disease to the first injection was 6 hours, 1; 12 hours, 1; 36 hours, 2; 2½ days, 1; 3 days, 1; 4 days, 1; 5 days, 2; 6 days, 4; 7 days, 4; 8 days, 1; 10 days, 1; 11 days, 1; a few hours before death, 2; not stated, 4.

It will be seen that twenty of the fatal cases did not receive the injection until after the third day. It is very generally admitted that the value of antitoxin rapidly declines after the third day of the disease.

The diagnosis was confirmed by culture in twenty of the fatal cases, but not in six. The number of injections given in the fatal cases was as follows: 1 in 17, 2 in 7, 3 in 1, 7 in 1.

The extent of the membrane was as follows: Tonsils in 1; tonsils and larynx in 1; pharynx and larynx in 2; larynx in 1; tonsils and pharynx in 2; tonsils, nose and pharynx in 3; tonsils, pharynx and larynx in 4; tonsils, nose, pharynx and larynx in 7; nose and pharynx in 1; not stated in 4. Intubation was performed in three and tracheotomy also in one of them.

The complications noted in the fatal cases were: Sepsis in 8; nephritis in 3; paralysis in 2; sepsis and scarlatina in 1; sepsis and nephritis in 1; sepsis, nephritis and paralysis in 1; bronchopneumonia in 1; strangulation in feeding in 1; not stated in 8.

The kinds of serum used in the fatal cases were: U. S. M. H.

S. in 8; Behring's in 7; Mulford's in 2; Roux's in 1; Fraser's in 1; not stated in 7.

The reporters commented upon their fatal cases as follows: One called his case malignant; two said theirs were hopeless when the injection was given; one said no effect was noted; five that the serum was used too late; two that the membrane rapidly disappeared; one that the case was of marked severity; five that the patients were moribund; one that temporary improvement followed the injection; and one that the patient was strangulated with milk. Seven offered no comments.

Treatment.—In almost all the diet was restricted to liquids, the preference being given to milk. The drugs used in the order of frequency were: Ferric chloride in 51; stimulants in 37; strychnia in 16; mercuric chloride in 15; quinia in 14; mercurious chloride in 4; digitalis in 2.

In a vast majority of cases internal medicine was stopped as soon as the serum was injected, with the exception of stimulants, strychnia and digitalis.

The local treatment appears not to have been neglected, as the following table shows. Hydrogen peroxide was used in 51 cases; potassium chlorate in 11; antiseptic sprays in 7; trikresol in 6; borolyptol in 5; listerine in 1; creolin in 1; vapor of quicklime in 1.

Antitoxin.—The kind of antitoxin used in each case is as follows: U. S. Marine Hospital Service in 66 cases; Behring's in 64; Roux's in 4; Mulford's in 5; Parke, Davis & Co. in 4; Aronson's in 2; Fraser's in 2; not stated in 30; total 177. One patient received Behring's at one time and Roux's the next, which accounts for the extra one.

The following are some of the comments of physicians upon the cases which recovered: Forty-two said the antitoxin "acted well;" thirty-two that its "action was marvelous;" ten that the "cases were mild;" two that it "was excellent;" one that it was "a most valuable adjuvant;" one that "improvement was rapid;" and one that "it had a marked effect." On the other hand, four said "it had no apparent effect;" one that "the case would have recovered under ordinary treatment;" and one that "it was given upon the demand of the parents, but the case would have recovered without it." Fifty-seven made no comments upon the remedy.

1 DUPONT CIRCLE.

THE CAUSE OF SUDDEN DEATH AFTER ANTITOXIN INJECTIONS.

BY A. SEIBERT, M.D.,

Professor of Pediatrics, New York Polyclinic,

AND F. SCHWYZER, M.D.,

Pathologist to the German Hospital.

In attempting to find the cause of sudden death after a hypodermic injection of antitoxic serum, all arguments point to the fact that death is caused by a something introduced under the skin of these patients, and not by shock. The labored respiration, the cyanosis, and the subsequent cessation of breathing reported in these cases give the clear picture of death from suffocation.

The second conclusion arrived at *a priori* is that this something injected under the skin could have exerted its deleterious action only by entering the circulation.

These conclusions led one of us (Seibert) to devise a series of experiments on animals, to determine as nearly as possible the conditions under which an injection of antitoxin could prove fatal.

The first possibility to be thought of was that the serum itself might do the killing either by entering the circulation too rapidly and in too large a quantity by an opened vein, or by poisonous substances having formed in the antitoxin in consequence of decomposition. Therefore the first series of experiments had to determine:

1. Whether large quantities of *fresh* antidiphtheritic serum brought directly into the blood current of an animal could produce unfavorable symptoms or death.

2. Whether large quantities of *old* antitoxin could cause trouble when used in this manner.

The second possibility to be thought of as a factor in causing sudden death after an antitoxin injection is the *carbolic acid*, said to be used in preserving the antidiphtheritic serum. Here it would be necessary to work with exact quantities and to determine how large a quantity of a given carbolic-acid solution could cause unfavorable symptoms and death. Beginning to

experiment with small quantities and weak solutions, and gradually increasing the amount and strength, would ultimately lead to the desired result.

The third possible factor as a cause of sudden death after the use of antitoxin is *air*, a quantity of which is found with the fluid in every syringe used for hypodermic injections. Here again it was necessary to first determine if air brought directly into the blood-current in limited quantities could cause pathological phenomena, then to find the amount of air necessary to cause disturbances or death, and at last to ascertain under what characteristic pathological and clinical manifestations such results would show themselves.

If animals could be killed with antidiphtheritic serum, the following second series of experiments with carbolic-acid solution would show whether this drug gave similar symptoms on being injected in the same manner, and, if not, would prove that as yet unknown powerful poisons had formed in the serum, and then old serum would be likely to be the most poisonous.

The intended experiments with carbolic-acid solutions would not alone prove the serum to be either free from or filled with a poisonous quantity of carbolic acid, but also would prove whether or not the symptoms caused by these injections were analogous to the ones found in patients dying after antitoxin injections. If at last it was shown by the first two series of experiments that animal life could not be quickly destroyed by either large quantities of antitoxic serum or by reasonably proportionate quantities of adequate carbolic-acid solutions, there would only remain to determine how small a quantity of air injected into a vein would suffice to cause the well-known death of air-embolism.

As it was clear that, whether serum, carbolic acid, or air caused death in these cases, the contents of the syringe after a hypodermic injection could reach the heart quickest by a vein, this channel was chosen to bring the antitoxin and its possible by-mixtures into the blood current.

After outlining this plan of action, the second one of the authors of this report (Schwyzer) was requested to take part in this investigation, so as to perform the laboratory work himself, bat always assisted by and in co-operation with the other author.

All the following experiments were made by means of intravenous injections, executed through Luer's blunt injection

cannula, filled with sulphate of magnesium (to prevent coagulation of blood), shut off by means of a stopcock, and tied firmly into the internal jugular vein by two ligatures.

From the start we confined our observations to the changes brought about in the animals during and immediately after the injections, and pronounced every animal unchanged in case its respiration and heart action were not altered during the twenty to thirty minutes after an injection. The action of the heart we observed in most animals by direct inspection of this organ before, during, and after each injection, through a window cut into the thorax by the resection of two ribs. Besides, we observed the heart's action by the stethoscope, and so could readily judge as to rhythm and strength.

Every animal was first narcotized with a mixture of chloroform, alcohol, and ether, then tied down upon its back, and then the throat and the thorax were exposed by removing the skin by an incision along the median line from the chin down to the diaphragm. Thus we were enabled to perceive any change in the circulation, especially as to a possible cyanosis. Before making an injection the effects of the narcotic were allowed to disappear so far that the corneal reflexes could be elicited. In no case was the narcotic continued after this response.

I. ANTITOXIN INJECTIONS.

First Experiment. — Full-grown guinea-pig. Four cubic centimetres of Behring's antitoxin No. 2, containing four hundred antitoxic units, filled December 20, 1895, at a temperature of 95° F., were injected rapidly into the right internal jugular vein.

Result.—Respiration and heart's action slightly accelerated (from 20 and 120 to 30 and 130 respectively), but returning to the normal within a minute.

Second Experiment.—Full-grown guinea-pig. Four cubic centimetres of a physiological salt solution were injected.

Result.—Identical with that of experiment No. 1.

Third Experiment.—Full-grown guinea-pig. Five cubic centimetres of Behring's serum, equal to 500 units, were injected.

Result.—Animal remained normal.

Fourth Experiment.—Full-grown guinea-pig. Five cubic centimetres of Behring's serum, equal to 500 units, injected.

No reaction. After ten minutes another dose of 400 units was injected.

Result.—Animal remained perfectly normal.

Fifth Experiment.—Full-grown guinea-pig. Five cubic centimetres of Behring's serum, equal to 500 units, filled December 23, 1894, injected, and two minutes later another dose of four cubic centimetres of the same.

Result.—No reaction whatsoever.

Sixth Experiment.—Full-grown rabbit. Ten cubic centimetres of Behring's serum, equal to 1,500 units, were injected within thirty seconds.

Result.—No reaction whatsoever.

II. CARBOLIC-ACID INJECTIONS.

Behring's antitoxic serum is supposed to contain a half per cent. of carbolic acid, so that five cubic centimetres would represent 0.025 of phenol. Our tests have been made with much stronger solutions.

First Experiment.—Full-grown guinea-pig. One cubic centimetre of one per cent. carbolic acid in physiological salt solution injected.

Result.—Immediately after the injection (two to three seconds), slight convulsive movements of the extremities, persisting during ten seconds; after this, respiration and heart normal. Within the following fifteen minutes three more doses of this one per cent. solution were injected at intervals of five minutes, resulting each time in the same slight, quickly disappearing twitchings. During the twenty minutes following these four injections (containing in all 0.04 of phenol) the animal remained perfectly normal.

Second Experiment.—Full-grown guinea-pig. Four cubic centimetres of one per cent. carbolic acid in physiological salt solution injected.

Result.—Violent convulsions, with marked cyanosis, accelerated respiration and heart's action (the beats too numerous to count) began immediately and persisted for twenty-five seconds, then there was an occasional twitching, and apparently the normal condition one minute later.

Third Experiment.—Full-grown rabbit. One cubic centimetre of a one per cent. carbolic solution was injected.

Result.—Slight occasional twitchings, five during the first fifteen seconds after the injection; normal after one minute.

Fourth Experiment.—Full-grown rabbit. Two cubic centimetres of a one per cent. carbolic solution injected.

Result.—Violent convulsions, with marked cyanosis immediately after injection, persisting for thirty seconds; then an occasional twitching; normal after three minutes.

Fifth Experiment.—Full-grown rabbit. One cubic centimetre of a two per cent. carbolic solution was injected.

Result.—Immediate violent tetanic convulsions, with cyanosis, persisting for thirty-five seconds; normal after one minute.

Sixth Experiment.—Full-grown rabbit. Two cubic centimetres of a one per cent. carbolic solution injected.

Result.—Convulsions during thirty seconds following the injection; normal after two minutes.

Seventh Experiment.—Full-grown rabbit. One cubic centimetre of a one per cent. carbolic solution injected twice at intervals of five minutes.

Result.—Slight convulsive twitchings, then normal.

Remarks.—In these experiments we injected from 0.01 to 0.04 of phenol into the jugular vein, and in every instance elicited a reaction perfectly identical in aspect, only varying in intensity and corresponding to the dose of carbolic acid. This reaction could not be due to simple mechanical irritation, as four cubic centimetres of a physiological salt solution have never produced a similar effect. As soon as the carbolic solution reached the heart (in two to three seconds) the convulsions began in each animal over the whole body, therefore long before the phenol had passed through the lung and could have reached the nervous system. This proves that these carbolic convulsions are most probably of a reflex character—*i. e.*, the phenol doubtless causes irritation of centripetal nerve fibres along the inner surface of the vein and of the heart. In the human body this irritation would be very slight, for even a small child has very much more blood than a guinea-pig or a rabbit, and therefore the percentage of carbolic acid would be proportionately smaller. We could distinctly observe that the effect of the carbolic injections in all of our experiments was much more dependent upon the concentration than upon the quantity of the solution injected, as is best seen by comparing the fifth and sixth experiments.

III. INTRAVENOUS INJECTIONS OF AIR.

That air brought into the venous circulation can prove fatal is well-known, but as yet the smallest fatal dose has (to our knowledge) not yet been determined. We have succeeded in finding this for the kind of animals used in the following experiments. While fully appreciating the difficulty in drawing conclusions from the animal to the human body, a certain analogy might be permitted, after comparing the capacity of the heart in guinea-pigs, rabbits, and children.

First Experiment.—Full-grown guinea-pig. Four cubic centimetres of air were slowly injected into the jugular vein.

Result.—Complete cessation of respiration within thirty seconds, after five jerky inspirations. Heart contractions were visible for five minutes, growing weaker continually.

Necropsy.—All blood-vessels leading to and from the heart were first ligated. Air was visible in the vena cava. The right ventricle and auricle were enormously dilated. There was air in the pulmonary artery; no air in the pulmonary veins. The heart floated on water. On opening the heart under water the right auricle and right ventricle were found full of air. The left auricle and ventricle were decidedly contracted. Neither contained any air and but little blood, hardly three-quarters of a cubic centimetre in all. All the veins contained much blood. The lungs appeared very pale and on section seemed to contain less blood than usual.

Second Experiment.—Full-grown guinea-pig. One cubic centimetre of air and four cubic centimetres of Behring's antitoxin (equal to 400 units) were injected together, the air in the middle of the fluid.

Result.—Marked dyspnœa and cyanosis within five seconds, respiration less and less frequent and more and more superficial. The heart beat at first over two hundred times a minute. Air in the heart could be distinctly heard through the stethoscope, like crepitant râles. Cessation of breathing within five minutes.

Autopsy.—After seven minutes slight heart contractions were still visible. After ligation of blood-vessels, the heart floated on water. Three-fourths of a cubic centimetre of air was caught under water from the right auricle and one-fourth of a cubic centimetre of air from the right ventricle, in a graduated tube. There was air in the pulmonary artery; no air in left heart, and very little blood. Lungs pale.

Third Experiment.—Full-grown guinea-pig. A quarter of a cubic centimetre of air was slowly injected by means of a screw syringe.

Result.—Respiration became slow and jerky, and cyanosis appeared. After two minutes the animal was apparently in a normal condition. Another dose of half a cubic centimetre of air brought forth similar symptoms, but more marked in intensity. After three minutes the animal was again normal. Then another half cubic centimetre of air was given with the same result. Air could be heard in the heart through the stethoscope. After another quarter of a cubic centimetre of air had been injected there was sudden respiratory paralysis.

Autopsy.—The same appearances were found as in the first experiment.

Fourth Experiment.—Full-grown guinea-pig. Every five minutes half a cubic centimetre of air was injected.

Result.—Sudden death after the third dose by cessation of respiration. A change of the heart's action from the normal 120, to over 200 beats a minute after the first half cubic centimetre could be distinctly seen, as well as the complete cessation of ventricular contraction on the third dose.

Autopsy.—The same appearances.

Fifth Experiment.—Full-grown rabbit. Two cubic centimetres of air in one dose. Death in five minutes.

Sixth Experiment.—Full-grown rabbit. Two cubic centimetres of air in one dose. Death in five minutes.

Seventh Experiment.—Full-grown rabbit. Two cubic centimetres of air in one dose. Death in five minutes.

Eighth Experiment—Full-grown rabbit. Two cubic centimetres of air in one dose. Death in five minutes.

Autopsies.—The appearances were the same as in those of the other animals.

Ninth Experiment.—Full-grown rabbit killed quickly by chloroform.

Autopsy.—Diastolic condition of the heart, excepting the left ventricle, which was slightly contracted. The blood-vessels leading to and from the heart were carefully ligated without pressure upon the organ. By first weighing the full heart, then emptying one chamber after the other and weighing after each opening, the following figures were obtained:

The full heart weighed 12.38 grammes; the right auricle con-

tained 0.92 grammes of blood; the right ventricle contained 2.12 grammes of blood; the left auricle contained 0.74 grammes of blood; the left ventricle contained 0.62 grammes of blood.

If we take the specific gravity of the blood at 1.060, to simplify calculations, then we shall have to deduct about six per cent. of the contents of the heart, so that in reality the right auricle would hold but 0.88 and the right ventricle but two cubic centimetres. The fatal dose of air injected into the vein of a rabbit appears to be two cubic centimetres—about half the capacity of the whole heart. It is similar in the guinea-pig, the heart of which is of about half the size of that of the rabbit, for here one cubic centimetre is the fatal dose.

If a large quantity of air is injected at once (one to two cubic centimetres in a guinea-pig and two to four cubic centimetres in a rabbit), then the right heart is simply filled by air, the circulation therein ceases, as it would require an enormous pressure to replace the blood in the capillaries of the lung by air, on account of the adhesive affinity of this fluid to the inner surface of these minute blood-vessels, and so death ensues very rapidly. If, on the other hand, a smaller quantity of air is injected (say thirty to sixty per cent. of the cubic capacity of the right heart), then air and blood have sufficient space by the side of each other, and the air, being of lesser weight, will collect in the upper recesses of the heart chambers, and death will result slowly through gradual insufficiency of the heart muscle to overcome the passive pressure of the air, together with the accompanying insufficient oxidation of the blood. In some cases a sufficient quantity of air may be pressed by the heart into the pulmonary artery to suddenly stop all blood supply to the lung.

Résumé.—In reviewing the foregoing experiments we may make the following statements:

1. Large quantities of fresh and of old antitoxic serum injected quickly into the venous circulation of guinea-pigs and rabbits did not cause the slightest reaction.

2. Carbolic acid in one and two per cent. solutions injected in from one to four cubic centimetre doses in the same animals invariably caused characteristic convulsions immediately, differing only in intensity and duration, proportionate to the quantity and concentration of the solution.

2. Air injected into the venous circulation of such animals invariably caused difficult respiration, rapid increase in the heart's

action, cyanosis, and ultimately death. The intensity and rapidity with which these symptoms appeared were in direct proportion to the amount of air injected into the vein at one time in so far that a larger quantity would cause death quicker than a smaller one; a smaller one injected quickly and at once would kill more speedily than a larger one injected slowly and at given intervals. A smaller quantity of air might cause speedy death (in five to ten seconds) if passed at once through the right heart into the pulmonary artery, causing complete occlusion, and a larger quantity remaining in the heart might not prove fatal for from five to twenty minutes.

Conclusions.—1. Antitoxic serum does not seem to be capable of causing threatening symptoms and speedy death, even when brought quickly into the blood current in very large doses.

2. The carbolic acid used in preserving the antidiphtheritic serum must be in such a weak solution as to be entirely unable to cause the characteristic carbolic convulsions produced in every one of our second series of experiments. The absence of these convulsions in the cases of sudden death in patients, the entirely different group of symptoms reported in them, and the fact that guinea-pigs and rabbits will survive even very large and concentrated doses of carbolic acid injected into a vein, lead us to discard the possibility of this drug having caused the reported deaths.

3. Even very small quantities of air will cause severe disturbances and ultimate cessation of breathing in every animal experimented upon. These disturbances are entirely analogous to the symptoms reported as preceding the sudden deaths after antitoxin injections. Air is found alongside of the fluid in every syringe used for hypodermic injections, and being pressed under the skin with the fluid may readily come in contact with a punctured cutaneous vein and so may enter the blood-vessel and the right heart, even before the serum has been absorbed.

In view of these facts and of our experiments, we here express our firm opinion that the sudden deaths reported after antitoxin injections were caused by injected air and not by the antidiphtheritic serum.

A CASE OF DIPHTHERIA SHOWING RAPIDLY FAVORABLE RESULT FROM THE USE OF ANTITOXIN.*

BY FREDERICK A. PACKARD, M.D.,

Visiting Physician to the Children's and to the Philadelphia Hospitals; Instructor in Physical Diagnosis, University of Pennsylvania, Philadelphia.

On November 24, 1895, I was asked to see Dr. W., with whom I had recently seen a severe case of diphtheria, in consultation. I saw him at 10 A.M., and obtained from him the following history: On November 20th he felt some sore throat, but this soon subsided and he had no subjective symptoms of angina until the evening of the day before I saw him. During the night he had been feverish, and early in the morning noticed a white patch on the right side of the throat. He had a considerable amount of lassitude.

When I saw him his face was flushed, his tongue coated, the expression heavy, pulse 96, temperature 102.3°. On the right posterior pillar and in the post-tonsillar groove there was a rather diffuse, thin, grayish-white deposit. The throat was so irritable that it was impossible to even touch the exudate without setting up such spasm in the tongue and pharyngeal muscles that it was impossible to discover the depth of the exudate. The whole surface of the fauces was of a brilliant red color. A swab and culture-tube were, however, inoculated with some difficulty. Heart and lungs were negative. Urinary examination negative. He was given calomel, one-sixth of a grain every two hours, and was given a local application consisting of corrosive sublimate 1 part, tincture ferri chlor. 500 parts, water 4,000 parts, to be used every two hours.

At 5.30 P.M. the temperature was 102⅔°, the pulse 88. There was decided general aching. He was given sodium salicylate, ten grains every three hours, and the calomel was discontinued. The nose was cleansed with Dobell's solution every four hours. It was impossible for any one save himself to make local applications to the throat owing to the reflex spasm started by even the approach of the tongue-depressor toward the lips. Fortunately, he could effectually apply the solution by the aid of a hand-mirror.

* Read before the American Pediatric Society, May 26, 1896.

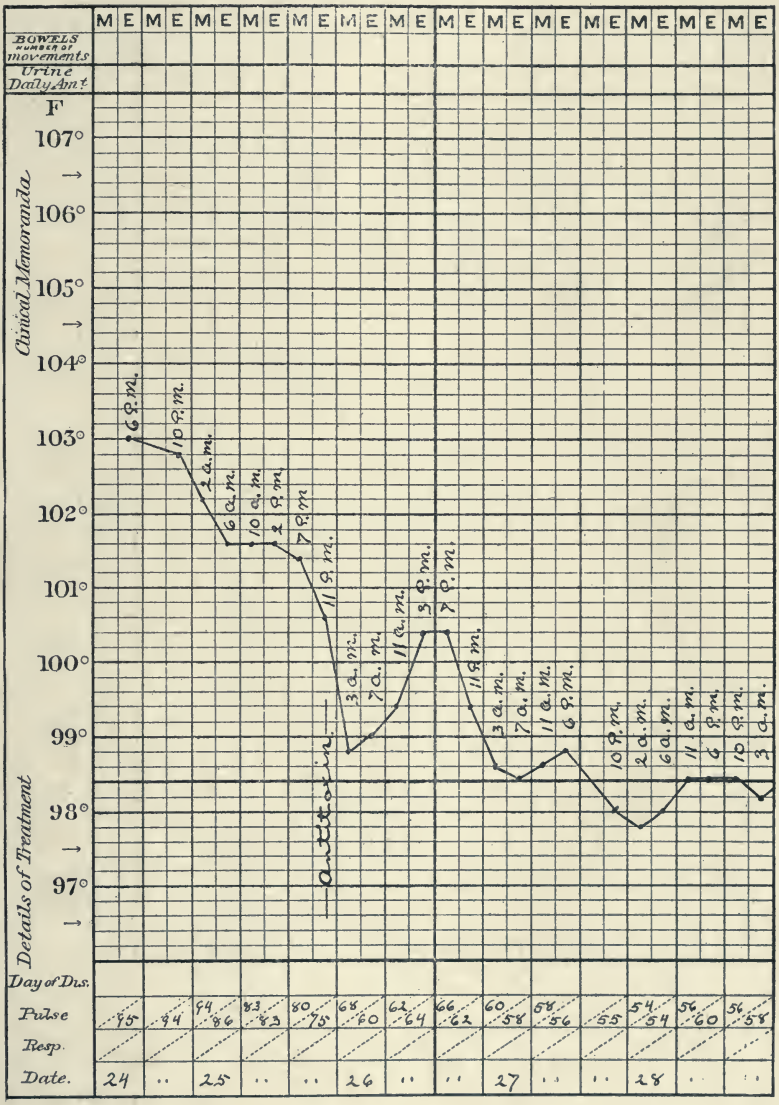
On November 25, the bacteriological department of the Board of Health reported the presence of "doubtful Klebs-Löffler bacilli." There was a linear grayish-black, slough-like exudate on the posterior pillar of the left side, and the right tonsil was much swollen and had grayish, isolated patches on its surface. The temperature was 101⅘°, the pulse 83. A small tender gland could be felt at each angle of the jaw. The urine negative. At 7 P.M. the temperature was 101°, the pulse 80. There was a slight extension of the upper patch on the right tonsil and on the left side, the exudate was decidedly more extensive than in the morning. Owing to the difficulty of making local applications, and also on general principles, 10 cc. (1,000 units) of Mulford's antitoxin were injected beneath the skin in the interscapular region at about 9 P.M. At 11 P.M. the temperature had fallen to 100⅗°; at 12.30 A.M. profuse sweating occurred; at 3 A.M. the temperature was 98⅘°.

When I saw him at 11 A.M. on the morning of the 26th, his expression was much brighter, his tongue clean, the membrane was quite loose and pultaceous on the left side, and there was only one small patch on the right tonsil. The general condition was much improved and he felt much better than at any time since his illness began. The report from a second culture, made on the 25th, was positive as to the presence of Klebs-Löffler bacilli.

On the afternoon the temperature went up to 100⅖°, but soon fell to normal. From this date nothing of interest occurred. The temperature remained normal, the pulse slow, sometimes being found at 56 per minute, but of good force and volume. The membrane rapidly came away, so that by the morning of November 28th, the throat was perfectly normal. After the membrane began to separate the corrosive sublimate and iron application was discontinued, and the throat was sprayed every two hours with equal parts of peroxide of hydrogen (15 vol. solution) and listerine.

The general condition steadily improved, and on the thirtieth of the month the patient sat up in a chair for an hour without fatigue. On the 5th of December the throat was free of bacilli, and he was taken out of quarantine. No sequelæ occurred, and the doctor was able to resume his practice after a precautionary visit of a week to the sea-shore.

The whole duration of his disease can be placed at five days



TEMPERATURE CHART.

the temperature was elevated for two and a half days, bacilli disappeared from the throat in twelve days, and the patient was able to be about the house in less than two weeks from the first beginning of his attack.

The change in the general and local condition within a few hours after the injection of antitoxin was too marked to leave room for doubt as to the specific benefit of the use of the remedy. Prior to its use the local process was rapidly extending, and local remedies could be applied but imperfectly. Immediately after its use the exudate softened and loosened, and while local applications were still attempted, it is very doubtful whether they were of any value.

110 SOUTH EIGHTEENTH STREET.

DISCUSSION ON ANTITOXIN.

DR. T. M. ROTCH.—This report is so excellent and exhaustive that it seems that there is hardly room for discussion. I merely speak of a coincidence, for it is rather remarkable Dr. McCollom in his hospital cases has presented almost exactly the same percentage, 12.5, while Dr. Holt gives 12.3. I think that is very interesting, and shows how exact the Committee's report is. These cases in the diphtheria ward under Dr. McCollom's supervision, who is an expert, were very carefully observed. In regard to the danger of antitoxin, every child who comes to the Children's Hospital of Boston, whether he has pneumonia or arthritis or diphtheria, receives antitoxin. This has been done in 700 or 800 cases, without any bad results whatever.

DR. AUGUSTUS CAILLE.—I think we have spent a very profitable morning. I think it is recognized that the report of the various hospitals as we have had them have told us very little. It is true that we have noticed a reduction in the mortality of 10 to 20 per cent. as compared with former years, when the death rate was low. This reduction is not large, and it is generally stated that the true test is the test which the Pediatric Society has made and reported on. Among 623 cases from private practice recently reported, the death rate in cases treated with antitoxin was reduced to about 7.

Our Committee's report, that the death rate will not exceed 5 per cent. if the cases are treated in time, is of great importance, and will certainly sway public opinion in the right direction. As regards the experiments of Dr. Seibert, they are interesting, and seem to point to the danger of injecting air. If so small a quantity will do injury, it is necessary to be absolutely careful in connection with injections.

My experience is fully in accord with what has been reported in the investigation. I have seen about sixty cases of diphtheria treated with antitoxin; thirty-five of these are incorporated in this report; the others are reported by other colleagues with whom I have seen cases, and the evidence is favorable. The most interesting point is the effect of antitoxin in diphtheritic stenosis, which the Committee has so carefully analyzed. I had a mortality of 70 per cent. before the use of antitoxin. In the cases treated with antitoxin, ten recovered and one died, and the one that died was one year old. I think he would have lived if the antitoxin had been given in time. It was given a day after the intubation.

DR. WM. P. NORTHRUP.—Can Dr. Seibert recall the manner of death of Miss Valentine, a case which received much attention about a year ago?

DR. SEIBERT.—She died within ten minutes of the injection. There was cyanosis and difficult respiration, and ultimately convulsions.

DR. NORTHRUP.—This young lady was in excellent condition, sixteen years old. It was tonsillar diphtheria. A thousand units of antitoxin were injected, and in ten minutes she became cyanotic, and died in convulsions. Those are the only facts reported by Kortright to the Committee.

DR. SEIBERT.—Dr. Langerhan reported that he injected his own child. He is a pathologist, and the injection was given as a prophylactic, and the child died within a few minutes after the injection. The autopsy showed nothing. If the air embolism has advanced further than the pulmonary artery, it is practically impossible to find it, that is to say, unless you are well trained to look for it. One of the difficulties in these experiments is in making the autopsy carefully. It is evident if the cause of death in the cases reported had been carbolic acid in concentrated solution, it would have caused convulsions within two or three seconds—the instant the carbolic acid reached the heart. In another case that is reported, the symptoms were difficult respiration, cyanosis, and ultimate convulsions. This suggests an air embolism. We refrained, as you notice, from making any suggestions regarding the amount of air required to produce death. My impression from what I have seen is that animals may be killed by air in small quantities. To my mind, ½ c.c. getting into the right spot, will suffice to kill very suddenly; that amount might stay in the heart, say five minutes, and then suddenly be pushed along. We made experiments with the body of the animals hanging down, and with the head downward. We thought we could change the rapidity with which the air would get into the pulmonary artery. Sometimes we thought we succeeded, and sometimes we did not. We left that out entirely for that reason, from the report.

In some animals, guinea pigs and rabbits for instance, we could gradually accumulate a large quantity of air, and then suddenly a small quantity would enter the pulmonary artery and shut off the breathing.

Dr. Wm. Osler.—Serious results have been known to follow injections of morphia. Hemiplegia even has followed a hypodermic injection of small size.

Dr. Henry Lafleur.—Anybody who has done work in anatomy knows that it is very difficult to strike a blood vessel with a needle. It is necessary to use forceps, and then one often fails. I think it is stretching a point to account for sudden deaths in that way.

Dr. L. Emmett Holt.—The third case of sudden death was published in the *New York Medical Journal* three weeks ago. A prophylactic dose was given. The injection was made in the back between the shoulders, and the girl was standing up.

Dr. James C. Wilson.—Sudden death has occurred from puncture of the chest.

Dr. Leroy M. Yale.—I had the misfortune once to get that result. The needle had hardly entered the chest when the patient succumbed.

Dr. John Dorning reported serious results which followed hypodermic injections in two cases, no air, he was certain, being injected.

Dr. Samuel S. Adams.—It seems to me that we cannot cast aside the influence of carbolic acid in producing sudden death, since we know there are individuals who are peculiarly susceptible to the drug. I have reported a case to this Society, and have recently seen a child who was poisoned by one-eighth of a drop.

So far as air is concerned, if that were the only factor in the causation of death many more people would certainly die from hypodermic injections. We know the average hypodermic syringe contains air, and physicians are often careless in its use. I agree with Dr. Lafleur that it requires some dexterity to inject a vein.

Dr. Northrup referred to two patients who had taken carbolic acid in large doses, for suicidal purposes. They both came into the hospital nearly asphyxiated, and both were resuscitated by artificial respiration by the O'Dwyer tube. Dr. O'Dwyer believed that the carbolic acid acted on the nerve centers to cause paralysis.

Dr. Booker.—In Dr. Kenyon's report upon the mortality of diphtheria in this country since the introduction of antitoxin, Baltimore is named as one of the cities in which the mortality of the disease has not been reduced. As this city was supposed be especially benefited by the valuable and convincing report on

the success of antitoxin, made by Professor Welch last year, it has been argued by some that the failure of the Baltimore statistics to show a diminution in the death rate of diphtheria, speaks rather strongly against antitoxin. So greatly at variance is the report of Dr. Kenyon from the opinion expressed by the physicians of Baltimore, who have used antitoxin in their practice, that I made special effort in collecting the cases for Dr. Holt to find the proper explanation for the unexpected statement of Dr. Kenyon.

I have seen many physicians representing different sections of Baltimore, and I am convinced from the information received from them, that a list of the great majority of cases in which antitoxin has been used, was forwarded to Dr. Holt. I do not remember the exact death rate in these cases, but it is certainly very low. I have heard but one opinion upon antitoxin from these physicians, that it is a rare thing to lose a case of diphtheria with the use of antitoxin.

Many physicians in Baltimore have not yet used antitoxin, and many did not begin its use until late in the winter. One physician who gave a list of three cases of diphtheria in which he had used antitoxin successfully, stated that he had not used it in twenty cases, and of these he had lost about 40 per cent.

The physicians of Baltimore often forget to report cases of diphtheria that end in recovery, and the proportion of cases that recover, and are never reported, is doubtless much greater than is suspected.

Dr. Charles W. Townsend.—How large an amount of air would be required to kill a girl of fifteen, supposing the heart to be in proportion to the size of a rabbit's heart? If 2 c.c. will cause death in a rabbit how much would be required to kill a fifteen-year-old girl?

Dr. J. Henry Fruitnight reported a striking result following the injection of antitoxin in a serious case.

Dr. Floyd M. Crandall.—In considering paralysis and other sequelæ, we must take into consideration the large proportion of recoveries. Assuming that antitoxin reduces the death rate, we should have a larger number of sequelæ because of the larger number of survivors. The conclusions derived from personal experience are as valuable as those derived from statistics. It is true that many errors in prognosis are made in diphtheria, but men of experience learn the disease and its peculiarities so thoroughly that their opinions regarding the merits of any plan of treatment are of great value. The expressions of personal opinion recorded in the Committee's report add much to its weight.

Dr. A. H. Wentworth.—Dr. Seibert said it does not depend on the amount of air injected, but the quantity of air that reaches the pulmonary area. It might be but ¼ c.c.; if it reached the

right place and blocked the artery, death would result very quickly. So we cannot estimate the quantity of injected air required to kill.

Dr. Caille.—Physicians have been taught by the bacteriologists that diphtheria antitoxin would not neutralize streptococcus sepsis. I suppose this has been a stumbling block. It is the custom of New York men to wait for a report of the case, and by that time it is too late. We should lay stress on this point, not to wait for the culture, but inject at once.

Dr. Holt.—When the physician makes his culture he waits for the bacteriological diagnosis. He is at last positive of his diagnosis, but loses his patient. He should trust to clinical symptoms, and not wait for a bacteriological diagnosis.

Dr. J. P. Crozer Griffith.—With reference to the mortality in different cities, it is well known to members here that the mortality in Philadelphia has been decidedly higher than in New York. Formerly it was not the case. This is no doubt due to adverse reports published in Philadelphia, which have deterred men from using antitoxin, have delayed its use until the case became very serious.

Dr. Holt.—Chicago is the only place where a real test has been made of the effect of antitoxin on city mortality. In 1895, diphtheria was epidemic there, and I have seen a chart of fatal cases of diphtheria for that time. They gradually increased to November, remained about the same during November, December, and January, and then slowly declined. Something like this had started last Fall, but with greater severity than previously. Until the use of antitoxin was begun the death rate was higher than in the year previous. In October the epidemic was so violent that it threatened to close many of the public schools, and became a very serious municipal question. From New York they got large quantities of antitoxin, and organized a corps of inspectors to use it. Of course, all the cases in Chicago were not treated by antitoxin, but a very large number were, and a remarkable reduction of mortality followed its use.

Dr. Seibert.—The effect which air will have in causing death will depend somewhat upon the position of the animal. Any quantity of air sufficient to suddenly enter the pulmonary artery or one of its larger branches will be sufficient to cause death. It is not simply the amount of air which enters the heart, but the amount that can pass out of it as an embolus. It would not be necessary that the needle should puncture a vein to cause absorption. If injected close under the skin, or in tense tissue, the air would be compressed and under great tension. If a lacerated vein lay in the right position, the air might be readily forced into it after the needle was withdrawn.

NASAL FEEDING IN DIPHTHERIA.*

BY HENRY JACKSON, M.D.,

Assistant Visiting Physician of the Boston City Hospital.

For some six or seven years in the diphtheria wards of the Boston City Hospital, gavage or forced feeding has been employed in the treatment of such cases as could not otherwise be properly nourished. Most of these cases were patients who had laryngeal diphtheria, in whom the O'Dwyer intubation tube had been inserted; other cases I shall speak of later. The majority of the physicians in attendance were so much impressed with the advantages of the method that it was employed more and more, until it became practically the routine treatment in all cases where intubation was necessary.

Through the courtesy of Dr. J. H. McCollom, who is now the resident physician, permanently in charge of the new contagious wards, I am able to report his methods.

In every case where intubation is necessary the child is fed by liquid nourishment, practically milk, introduced by a catheter passed into the stomach through the nose. From September 1, 1895, to February 1, 1896, the new wards having been opened on September 1st, eighty-nine cases have been intubed, of whom fifty-six recovered, giving a mortality of 27 per cent. Of course all received antititoxin. This mortality is slightly lower than the average, 28.9 per cent., given by Dr. Welch as the result of his collective examinations of a large number of cases where intubation was employed in various hospitals. The cases reported from the City Hospital were all severe, intubation having been done in the vast majority of cases within twenty-four hours of admission to the hospital. It has been the experience at the City Hospital, as in other institutions, that where antitoxin was given soon after the larynx was invaded that no operative measures were necessary. Antitoxin checks the advance of the disease in the larynx, as we can see that it does in most of the pharyngeal cases where we can exactly localize the extent of the affected areas.

To quote from the paper of Drs. Prescott and Goldthwaite (*Boston Medical and Surgical Journal,* Vol. cxxv., page 694), intubation was first employed at the City Hospital on December 31, 1886. Since 1890 it has been practically the only operation, tracheotomy being done only when intubation fails, or when the

*Read before the American Pediatric Society, May 26, 1896.

soft parts above the larynx are so swollen as to render the intubation tube useless. In this paper, published in 1891, Drs. Prescott and Goldthwaite speak of the difficulty in feeding patients, recommending in some instances the giving of soft solids or using the postural method of Dr. Casselberry (*Chicago Medical Journal and Examiner*, October, 1888). To relieve thirst, which often was a very troublesome symptom, warm water enemata were given or cracked ice was allowed by the mouth.

Surely after intubation it is as necessary as in any other disease to support the child with the best possible nourishment. We have to deal with a disease accompanied by severe toxæmia, usually associated with secondary sepsis, and in addition the laryngeal tube, which mechanically renders feeding at best difficult and often in sufficient quantities impossible.

Throughout the world the great advantages of intubation over tracheotomy are appreciated and acknowledged, and in most hospitals tracheotomy is performed only if intubation has failed, or when the anatomical conditions are such as to render intubation insufficient to relieve the existing obstruction.

The drawback to intubation is the difficulty of feeding the patient. A few patients easily learn to take liquid food; in most it causes cough. Others may take food from a nursing bottle who cannot drink. If these methods fail we have recourse to the postural method of Dr. Casselberry, or the use of semi-solids as custards, etc. Nutritive enemata may be tried, but in many and perhaps most cases fail. In looking over the hospital records I found few cases in which rectal enemata proved satisfactory. It has been suggested to remove the tube to feed the child, but so far as I know no one approves of this method. Finally, feeding by the œsophageal tube.

The literature of the last procedure is, so far I can find out, scanty.

M. D'Heilly, in the *Gazette des Hopitaux* for May 3, 1888, in an article on intubation, gives the opinion that feeding, from the first, should be by means of an œsophageal tube.

Under the title "Gavage in the Treatment of Acute Diseases of Infants and Children," Dr. Holt contributes an important paper to the subject in hand, though the paper was not written to consider the after treatment of intubation. Dr. Holt in this paper advocates forced feeding in certain acute diseases of infancy, advising passing the catheter through the mouth rather than through

the nose, stating that in 400 cases it was rarely necessary to pass the tube through the nose. I take the liberty of quoting directly from Dr. Holt's paper, as in his description of his first case, severe pharyngeal diphtheria fed by the nose, he tersely lays down what I believe to be the most important underlying principle of the methods I am considering: "The child was being worn out by well-nigh fruitless efforts to get her to take even the smallest quantity of food and stimulants." Again, in a "very bad case of pharyngeal diphtheria," Dr. Holt says, "she was being worn out by constant teasing and forcing." Dr. Holt's paper advises gavage in the following cases:

1. Premature infants (chosen cases).
2. In some cases of obstinate vomiting.
3. In acute diseases when food is refused, either on account of the pain caused by the ingestion of food or on account of an utter distaste for food of any kind.
4. In certain cerebral cases.

In summing up, Dr. Holt says that in his opinion "rectal alimentation is very unsatisfactory, and in most cases an entire failure." (*Medical Record,* 1894, pages 5–24).

Dr. W. A. Morrison, of East Boston, published in the *Boston Medical and Surgical Journal* of February 7, 1895 (paper read in March, 1896), a paper entitled "The Value of the Stomach Tube in Feeding after Intubation, Based upon 28 Cases." These cases he reports from his private practice, all treated without the assistance of a trained nurse. Dr. Morrison considers the method as relatively easy and as undoubtedly a means of saving life in certain cases. Dr. Morrison concludes his paper with the details of a brilliant case, one of extreme exhaustion with post-diphtheritic paralysis which apparently prevented swallowing. The child was fed by a nasal tube for ten weeks and recovered. Twelve children recovered, giving a mortality of 57 per cent., nine of the sixteen cases dying within twenty-four hours. In a personal letter recently received from Dr. Morrison he adds that in debilitated cases he continues the treatment as long as called for.

Dr. C. M. Whitney of Boston, who has had extensive experience with intubation in private practice, writes me that he has tried the method suggested by Dr. Morrison. "I have tried this method several times in the last year and a half with perfect satisfaction in every case. I reserve it for those cases in which swallowing is difficult, in spite of the postural method of feeding which

is sufficient in half the cases. If it were possible I should use it in nearly every case, for it has the great advantage of permitting the placing in the stomach of a definite amount of food and medicine at fixed intervals with a minimum of discomfort to the patient. The tube passes with perfect ease, with very little discomfort to the patient, and in very few instances is there regurgitation of food."

Dr. C. B. Stevens, of Worcester, has reported in the *Boston Medical and Surgical Journal*, for April 25, 1895, a case of nasal feeding, and he allows me to quote from a paper which he is soon to publish in the same journal. "By the Casselberry method of feeding, the food is apt to run up into the posterior naso-pharynx and out of the nose, especially if there be extensive membrane in the throat. Whiskey and drugs especially irritate the child when so taken, and the child soon refuses to take all food in sufficient quantity." "The advantages of nasal feeding are that nothing gets into the trachea, that the naso-pharynx is not irritated by food and drugs, and that sufficient and known amounts of food and drugs can be given at regular intervals. The children who need food and stimulants most refuse them."

Dr. Stevens' cases are as follows (9 cases):

4 fed by postural method; 2 died.

4 fed by nasal tube; 1 died in twelve hours of broncho-pneumonia.

1 by enema; death in two hours.

Dr. Stevens also reports three cases of peritonsillar abscess, all of whom refused food on account of the pain in swallowing. They were fed with advantage by the nasal tube.

All of these cases were in private practice, and fed three times a day without the aid of a trained nurse. In the intervals between the feeding the children were refreshed by long and quiet sleep.

Methods adopted at the Boston City Hospital: The child is carefully pinned in a blanket so that it cannot move its arms; it lies on the back. Usually one nurse feeds the child. A glass funnel about four or five inches in diameter is attached to a Jaques soft rubber catheter with the intervention of a bit of plain glass tube. The catheter should be from 7 to 15 F., according to the age of the patient, the larger sizes being the more desirable. The well-oiled catheter is passed quickly but quietly along the floor of the nares. Just as it passes into the œsophagus there is usually slight resistance and gagging, otherwise no trouble is found. A small amount of water is allowed to flow

down, and, without allowing any air to enter, the milk and medicine are poured immediately into the funnel. At the end of the feeding again a little water is given.

Children are usually perfectly quiet as soon as the tube has been passed; and I have many times seen children take food in this way as quietly as from a nursing bottle. In withdrawing the catheter it should be pinched, that the few drops remaining may not flow out and irritate the pharynx.

In this way the child is fed every four hours, the following amount being given:

Water, 1 oz.; milk, 4 oz. to 6 oz.; brandy, ½ drachm to 2 drachms or more.

Digitalis, strychnia, or other drugs as indicated in the special case. Careful charts are kept of each case and I append a sample:

Child, age 5, with membrane in pharynx and laryngeal obstruction. was admitted at 1.30 A.M. Antitoxin given; intubed at once.

2 A.M., received milk, 4 oz.; brandy, 2 drachms; tincture of digitalis, 1 minim.

The child was then fed as stated every four hours with the following results:

First night, treatment mostly retained; first day. treatment mostly retained; second night, nearly all retained; second day, nearly all retained; third night, all retained; third day, mostly retained; fourth night, all retained; fourth day, mostly retained; fifth night, all retained; fifth day, tube extracted; bread and butter, milk, etc.

The patient recovered. This case was taken at random from a large number, and is a fair representative of the series.

Cases are so fed until the tube is removed; and non-laryngeal cases have in some instances been fed two or three weeks. I have already spoken of a case of Dr. Morrison's fed ten weeks.

The milk with medicine added must be on a table within easy reach of the nurse, and care must be exercised not to allow air to enter with the milk. The milk is taken and the patient is quietly resting in a very few minutes.

Advantages: A known and sufficient amount of food and stimulant is given.

The child is not disturbed as much as by constant and often ineffectual efforts to persuade it to take even a small amount of food.

In the intervals the child is not disturbed, and as it has received a good meal it usually sleeps quietly.

All of these arguments may be made in the case of private as of hospital patients. In the hospital the additional argument of the great saving of time is potent, as it is difficult to properly feed a child, even though it be willing to take food, unless we have the undivided attention of a nurse for each case.

As the child receives water in addition to its food, thirst, often a most distressing symptom, is rarely complained of. Introduction of milk by this method causes so little disturbance that a private patient of mine, not an intubation case, used to watch the milk run through the tube and say, "Katy wants to know where milk is going." Dr. McCollom tells me that in only one case was he prevented from using a nasal tube; in this case on two attempts the child stopped breathing and became cyanotic; he was unable to say whether it was due to some obstruction or due to reflex irritation. No ulceration of the nose has followed the use of the tube; no swelling from the mechanical irritation.

Occasionally the tube may pass out from the mouth on the first attempt, but apparently there is no danger that the tube will enter the trachea.

Aside from the cases of intubation, nasal feeding is employed in the contagious wards in two other classes.

First: In post-diphtheritic paralysis, where nutrition is interfered with either because a large amount of the food is regurgitated through the nose or in occasional cases where food is vomited as soon as taken.

Second: Nasal feeding is of great value in the important class of cases that refuse all nourishment. Food may be refused by the child, because swallowing causes so much pain, or because of an entire lack of any desire for food in severely prostrated children. During the last year six cases of malnutrition, the result of post-diphtheritic paralysis, and several cases that refused food have been fed by the nasal tube. All with satisfactory results. The latter class of cases is included in the diseases in which Dr. Holt advises forced feeding with the œsophageal tube.

Once in the stomach it seems as if we had won half the battle as far as food is concerned. This is shown by a chart which I happened to see. A child resisted vigorously the nasal feeding, coughed a good deal during the process; yet all the food was retained and the child recovered.

I wish to report briefly the notes of a private case of severe pharyngeal diphtheria in which I feel that nasal feeding added

much towards recovery. Child three years and nine months. Previously healthy.

December 30th. Prostration; temperature, 104°; pulse, 160; two slight convulsions; vomited; on right tonsil a few spots; culture taken which showed the presence of the Klebs-Löffler bacilli.

December 31st. Both tonsils, uvula and pillars of pharynx covered with thick gray membrane. During the day a skilful nurse succeeded in giving her only one ounce of food; in the evening four ounces by nasal tube. The pulse, which had been at times intermittent, improved, and she slept quietly, taking at intervals twelve ounces of milk.

January 1st. Fourteen ounces milk by nasal tube in divided amounts; nine by mouth.

On January 2d and 3d she was fed by the nasal tube, and took but little by the mouth. After each feeding the pulse improved and she slept quietly for several hours. She usually asked for a drink just after the tube was removed. After this there was a gradual improvement with final recovery.

I have had no experience with the introduction of an œsophageal tube through the mouth, but from my experience with adults I feel more inclined to continue the use of the nasal tube.

In closing I wish to express my thanks to Dr. J. H. McCollom for permission to report his statistics.

309 MARLBORO STREET.

DISCUSSION.

DR. L. EMMETT HOLT.—Dr. Jackson has referred to some of my cases. It seems to me that the great thing is the forced feeding; whether through the nose or through the mouth is secondary and a matter of convenience. Most of my patients had few teeth, and consequently the mouth has been the simpler way. In children who have teeth and can bite, it is, I think, the best practice to pass the tube through the nose. Any one who has watched children die from acute infectious disease, must be convinced how frequently death occurs from slow exhaustion, from the inability of the child to take and digest food. As to the frequency with which food should be introduced, I make the interval a little longer for older children, which makes the labor less. I think it is quite sufficient to feed four times in twenty-four hours. Ten ounces can be given as easily as four or five ounces.

Milk given under these circumstances should always be partly peptonized, and if for very young children it should be com-

pletely peptonized. In very young children the stomach should be washed every day because of the large amount of mucus. To remove it aids absorption. I have been struck with the great benefit of this method of forced feeding in young infants with pneumonia. I remember a case following influenza in a child of three months—an extremely severe case. The prostration was so great that the infant was kept alive for forty-eight hours by regular and systematic feeding. In another case the temperature was 106¾°. It was fed in this way for three weeks.

In the infectious diseases patients, if thus fed, are not worn out by efforts to get down a little food. A child is given sufficient food for one meal, and can then rest for some hours until the next feeding.

Dr. J. Henry Fruitnight.—In cases accompanied by coma, I think this method of feeding is very wise, and the only method to be relied on. The results are very satisfactory.

Dr. Wm. D. Booker.—It may seem strange that I should say that I have never employed nasal feeding in diphtheria. The subject in discussion is nasal feeding in diphtheria, and I shall therefore confine my remarks to diphtheria, and not refer to pneumonia. The question which arises in my mind has been for years, when I see a child with diphtheria, is it right to feed as often as in health? The next question is, will that child digest anything that is forcibly brought into the stomach?

Within the last two years, especially since I have had the aid of antitoxin, I have discarded all forced feeding. I have discarded all coaxing to feed. My principle has been, for eight or ten years, in every infectious disease in young children to give less food and to give it less often. Such children will digest less than in health, and it will take longer. For that reason I think it is absolutely wrong to give the same quantity as in health to a child suffering from diphtheria or any other infectious disease. The question has come to my mind, is it correct to put food into the stomach when a child does not want it? I believe in diphtheria that there is no necessity for it. The so-called heart-paralysis is not due to the want of food. It is simply due to the action of the toxin of the disease and nothing else.

I fully agree with Dr. Jackson and Dr. Holt that nasal feeding and forced feeding is advisable in some cases. I think in diphtheria it is unnecessary.

Dr. Charles G. Jennings.—I am accustomed to use nasal feeding as described by Dr. Jackson, in cases of grave diphtheria. At the time that the feeding is done we can use the catheter for any therapeutic purpose we may desire, for medicine, as well as food, may be introduced through it.

TAPPING THE VERTEBRAL CANAL.

Local Treatment for Tubercular Meningitis.

BY AUGUSTUS CAILLÉ, M.D.

Professor of Pediatrics, New York Post-Graduate Medical School and Hospital; Visiting Physician to the German Hospital, New York.

The subject under consideration has a special interest for me from the fact that as early as 1876 I had participated in a series of experiments on dead and living animals for the purpose of corroborating Quincke's discovery in 1872 of a free communication of the subarachnoid space of the brain and spinal cord. These experiments were made at the Wurzburg pathological laboratory and are laid down in an Inaugural Thesis by Buschbeck, Wurzburg, 1877. The possibility of diffusing colored liquids through the subarachnoid space of brain and spine has been firmly established for more than 25 years, and it may appear strange that a practical application of such knowledge should have hung fire for so long a period until the original experimenter, Quincke, in 1891 again drew attention to these facts and to the possibility of tapping the spinal canal in the lumbar region.

This tardiness in establishing local treatment for affections of the central nervous system is readily accounted for by the difference in pathological conception of to-day as compared with that of a quarter of a century ago.

Virchow's cellular pathology with one blow demolished the idle philosophical theorizing in medicine in vogue in the early part of this century, and gave us a solid foundation for more exact work, but the labors of Pasteur, Lister, Koch, and Behring have given us the real key to the hitherto mysterious biological

and pathological phenomena and have furnished the missing link which connects cell proliferation with the pathological phenomena of the living organism as we view it to-day, and gives stimulus and inducement for direct local antiseptic treatment.

Since presenting the subject of lumbar puncture of the subarachnoid space at the meeting of the American Pediatric Society in May, 1895, I have continued my investigations of the cerebro-spinal fluid as found in various pathological conditions, and have also made an attempt at direct local treatment of tubercular meningitis and the result following this line of treatment I beg to report in a brief manner. The work was conducted during my five months' term of service at the Babies' Wards of the Post-Graduate Hospital. The chemical and bacteriological examination by culture and microscope of the fluids was made by Dr. F. Schwyzer, Assistant Physician to the Babies' Wards, and also at the laboratory of the school in charge of Dr. Brooks.

The following tabulated list of cases will show the character of the cerebro-spinal fluid found in various conditions of disease. In most of the cases the fluid withdrawn was examined for sugar, albumin, and bacteria, and the clinical diagnosis was verified by autopsy in every case possible. The fluid was collected under strict antiseptic precautions by means of a syringe made entirely of glass and was transferred to a sterile glass tube and at once sealed up again in a flame.

In most of the cases the pressure of the fluid was such as to force out the piston of the syringe. In only two cases did a rise of temperature follow the puncture — no difficulty was encountered in puncturing and no anæsthetic was employed. In no case did the fluid aspirated contain blood, and no unpleasant after effects of any kind were noticed.

The first four cases in the list are the cases reported to this Society at our last meeting. Cases 1, 2, and 3 were typical cases of tubercular meningitis in which the bacilli were found. Specimen 4 was taken from a woman, thirty-five years of age, suffering with acute mania who, in the third week of her illness showed an elevation of temperature and symptoms of cortical irritation, particularly choreiform movements of the upper extremities. The liquid proved to be sterile but contained albumin and one-half of one per cent. of sugar. Meningitis was excluded and a favorable prognosis ventured which proved to be correct. The fluid from a case of cerebro-spinal meningitis (Case 5) contained

CASE.	SEX.	AGE.	CLINICAL DIAGNOSIS.	SUGAR.	ALBUMIN.	BACTERIA.	AUTOPSY AND REMARKS.
1	M.	3 y.	Tubercular Meningitis	. .	. .	Tubercle bacilli	. . . .
2	M.	4 y.	Tubercular Meningitis	. .	. .	Tubercle bacilli	Tuberc. Meningitis.
3	F.	2½ y.	Tubercular Meningitis	. .	. .	Tubercle bacilli	Reported June 15, 1895, in *N. Y. Medical Journal.*
4	F.	35 y.	Acute Mania	½ p.c.	Trace	Sterile fluid	. . . .
5	M.	5 y.	Cerebro-spinal Meningitis, Broncho-pneumonia	¼ p.c.	Trace	Pneumococcus	Recovery
6	M.	3 y.	Sarcoma of Kidney, Broncho-pneumonia	⅛ p.c.	. .	Pneumococcus	. . . .
7	F.	2 ½ y.	Tubercular Meningitis	. .	Trace	No bacilli found	. . . .
8	F.	1 y. 4 m.	Broncho-pneumonia, convulsions	¼ p.c.	Present	Tubercle bacilli	Tuberc. Meningitis, Catarrhal Pneumonia
9	F.	1 y.	Diphtheria of Pharynx	. .	. .	Staphylococcus pyogenes aureus	. . . .
10	M.	2½ y.	Pneumonia Dextra	. .	3 p.c.	Pneumococcus	. . . .
11	M.	2 y.	Acute Hydrocephalus	Trace	Trace	Sterile fluid	. . . .
12	M.	1½ y.	Acute Hydrocephalus	Trace	Trace	Sterile fluid	
13	M.	½ y.	Acute Eczema, Nephritis	. .	Trace	Streptococcus	Autopsy: Acute Nephritis
14	F.	⅓ y.	Hydrocephalus	. .	. .	Sterile fluid	. . . .
15	F.	6 y.	Tubercular Meningitis	. .	. .	No tubercle bacilli	Had apex pneumonia a year before
16	M.	1½ y.	Tubercular Meningitis	Trace	Trace	Tubercle bacilli found	Autopsy: Tuberculosis of meninges and lungs
17	M.	6 y.	Tumor of Brain	1/5 p c.	Trace	Sterile fluid	Cysto-sarcoma of brain
18	M.	8 m.	Diphtheria of Nose	. .	. .	Streptococcus	. . . .
19	F.	5 y.	Chorea	. .	. .	Sterile fluid	. . . .
20	F.	2 y.	Pertussis	. .	. .	Sterile fluid	. . . .
21	F.	4 y.	Tumor of Pons.	. .	. .	. . . .	Only a few drops of fluid obtained

pneumococci and the same form of bacteria were found in Case 6, a child which died from exhaustion in consequence of renal sarcoma with broncho-pneumonia. In Case 7 the tubercle bacilli were not found, which does not prove their absence however.

Case 8 presented the clinical picture of broncho-pneumonia and convulsions. Tubercle bacilli were found and an autopsy was secured.

Case 9, a girl one year old, who developed Klebs-Löffler diphtheria in the wards and subsequently died at the Willard Parker Hospital. In the spinal fluid of this case the staphylococcus pyogenes aureus was found and we must look upon this case as particularly interesting from a pathological standpoint.

In Case 10, a pneumonia with cerebral symptoms in a boy two and one-half years old. the liquor cerebro-spinalis contained three per cent. of albumin, also pneumococci.

Cases 11 and 12 were ordinary cases of hydrocephalus without special interest, but Case 13 is worthy of note from the fact that the streptococcus was found in the cerebro-spinal fluid of a child of five months which had been suffering from an ordinary universal eczema and died rather suddenly from acute nephritis. Here the diagnosis was verified by autopsy. The youngest child punctured was Case 14, a child four months of age with congenital hydrocephalus, and a thick fat skin which made puncture somewhat difficult, as it was not possible to feel the spinous processes of the vertebræ. I succeeded in getting into the spinal canal at the second attempt.

Case 15, a girl of six years, who died of tubercular meningitis; the bacilli were not found although considerable care was exercised in searching for them.

Case 16, tubercular meningitis, bacilli in the fluid; autopsy.

Case 17, cysto-sarcoma of the cortex of cerebrum in a boy six years old. The spinal fluid was sterile but contained one-fifth of one per cent. of sugar. The diagnosis was verified by autopsy.

Case 18, nasal diphtheria with general sepsis in an infant eight months old. Streptococci were here found in the fluid.

Case 19, a girl of five with uncomplicated chorea; sterile cerebro-spinal fluid.

Case 20, pertussis of moderate severity. In this case also the spinal fluid was free of bacteria.

Case 21, a girl four years old afflicted with a tumor of the

pons, syphilitic or tubercular. A puncture was made between the fourth and fifth lumbar vertebræ and also between the third and fourth vertebræ and only a few drops of a perfectly clear liquid were obtained, not a sufficient quantity for examination. In this case which is still under observation at the Babies' Wards the tumor is probably so located as to interfere with the free communication between the sub-arachnoid space of spine and brain.

A careful study of the reports on lumbar puncture of the sub-arachnoid space which have appeared up to date will certainly convince the most skeptical that Quincke's puncture is of positive value as a method of diagnosis. It is simple and usually easy of performance. When performed without narcosis the patient must be securely held in the proper position and great care must be exercised so as to avoid breaking the needle or injuring deep and important structures.

It will naturally occur to any one working in this line that a liquid may be just as readily injected into the spinal canal as it is removed therefrom, particularly after the pressure of the fluid has been diminished.

This I have done in two cases with a view of favorably influencing the course of an otherwise incurable tubercular meningitis and in hopes of gaining somewhat similar results as we obtain in the local treatment of tubercular peritonitis.

The first instance in which I attempted local treatment is Case 8 on our list. The patient, a girl of sixteen months, lying in convulsions for over twelve hours was first tapped and 20 c.c., of cerebro-spinal fluid were allowed to flow out. I then injected fifteen grains of sodium salicylate dissolved in 5 c. c., of water (sterilized). This procedure had no special noticeable effect upon the convulsions, pulse or temperature of the child. Death took place two days later and the autopsy performed by Dr. Ogden, our house surgeon, showed a soft œdematous brain, studded with tubercles and the lungs in a state of catarrhal pneumonia.

The second patient, a boy of eighteen months, was an advanced case of tubercular meningitis in coma, Case 16 of list. After removing 20 c.c. of fluid in which the specific bacilli were subsequently found, I injected five grains of iodoform suspended in 5 c.c. of sterilized water.

A rise in the frequency of the pulse was observed, otherwise nothing of importance. On the following day a second injec-

tion was made into the lumbar sub-arachnoid space. Death took place four days later. The autopsy performed by Dr. Ogden showed tubercles at the base and convexity of the brain and miliary tuberculosis of lungs and surface of spleen.

The two cases here cited prove to me that a more thorough washing of the sub-arachnoid space is necessary in order to make an impression upon a case of tubercular meningitis. At the next opportunity which presents itself I propose to lay bare the dura by removing a button of bone with the trephine and irrigate the subarachnoid space from a lumbar puncture upward through an opening in the dura. Irrigation by the shorter route through the lateral ventricles will probably not reach the convexity and will be inadequate.

753 MADISON AVENUE.

SOME EXPERIMENTAL WORK ON LUMBAR PUNCTURE OF THE SUBARACHNOID SPACE.

(From the Sear's Pathological Laboratory of the Harvard Medical School.)

BY A. H. WENTWORTH, M.D.,

Assistant in Diseases of Children in the Harvard Medical School; Out-Patient Physician to the Children's Hospital; Senior Assistant Physician to the Infants' Hospital, Boston.

The object of this paper is threefold:

First. To show that the withdrawal of fluid from the subarachnoid space by means of lumbar puncture is a harmless procedure.

Second. To show that the slightest cloudiness present at the time when the fluid is withdrawn, and caused by the presence of cells in the fluid, and the formation of fibrin in the fluid after it has stood for several hours, are pathognomonic of an inflammatory exudation in the meninges, and are never absent in cases of meningitis.

Third. To show that the normal fluid is absolutely clear and free from all cellular elements and fibrin.

I performed the operation of lumbar puncture for the first time in August, 1895, on a doubtful case of tubercular meningitis. A brief report of this case was published in the *Boston Medical and Surgical Journal* of December 12, 1895. After allowing five or six cubic centimetres of fluid to escape, the needle was withdrawn. Immediately following this the patient showed alarming symptoms. Her pulse rose to 250 beats in the minute (counted with a stethescope); she clutched at her hair; tossed herself about the bed, and uttered sharp cries. Her color was not good, and her extremities became cool. Subcutaneous injections of ether and brandy were given, and heaters were applied. After one-half to three-quarters of an hour the symptoms had passed away.

During the attack I felt considerable uneasiness because I was unprepared for such a result, and did not know but that it would terminate fatally. I now believe that the symptoms were due to headache, caused by the removal of fluid, and that her life was not endangered.

This patient did not have meningitis, and left the hospital shortly afterward, perfectly well.

At the time, I tried to explain the symptoms by assuming that the sudden alteration in intra-cranial pressure had caused them. In cases of meningitis there is an increase in the amount of fluid in the subarachnoid space, and the withdrawal of some of it tends to relieve the increased pressure temporarily. But if there were no increase in the amount of fluid, it appeared to me possible that the disturbance of the normal pressure might be a dangerous procedure. If this were so, the operation could not be considered so harmless as was generally supposed.

After several months' consideration I resolved to try some control experiments on normal cases in order to determine whether or not the operation was a dangerous one. The diagnostic value of puncture of the subarachnoid space is so evident that I considered myself justified in incurring some risk in order to settle the question of its danger. If it proved to be harmless, then one need not wait until a patient became moribund before resorting to it.

The differential diagnosis between the various forms of meningitis and many other diseases, especially in infants and children, frequently presents great difficulties and is often incorrectly made.

Any one who has had opportunities for observing infants and young children knows how variable their symptoms are, and with how much less certainty one can predict the reaction which will follow a given condition than in the case of an adult. For these reasons it is of the utmost importance to possess a reliable means of diagnosticating so common a disease as meningitis.

In this connection it appears to me to be of primary importance to diagnosticate the meningitis, and secondarily to determine the variety. It may not be superfluous to describe the methods of procedure and to allude to some of the contingencies which may occur.

The back of the patient was thoroughly cleaned from the last three ribs downward to the sacrum with alcohol, ether and a solution of corrosive sublimate, 1. to 2.000. It is hardly necessary to say that the operator's hands should be thoroughly cleaned and disinfected. The needle was boiled for ten minutes. An antitoxin needle is preferable to an ordinary hypodermic needle; it is less liable to break and has a larger lumen, besides

being somewhat longer. The one used on children over three years of age was four and one-half centimetres long, with a diameter of one and one-half milimetres. For infants under three a needle four centimetres long, with a diameter of one milimetre was used.

A syringe is never necessary, but it is well to have a sterile wire to pass through the needle in situ, in case the fluid does not run well.

Most of the punctures were made between the third and fourth lumber vertebræ, others were made between the second and third. All of the punctures were made with the patient lying on the right side, and it was found that the position of the patient was an important detail in rendering the operation difficult or easy. It is well to have the patient bent somewhat forward so as to separate the spinous processes, and what is of more importance, to have the spinal column presented directly toward the operator, so that the back forms an obtuse angle with the table or bed. This enables the operator to control the direction of the needle better, because he thrusts the needle directly forwards rather than from the side.

In some cases when this position of the patient was not carefully observed, it was found that the point of the needle crossed the median line toward the left and struck on the bone.

All of the work was done on infants and children, and on them it is not difficult to find the third or fourth lumbar vertebræ without trying to remember various lines and landmarks. The free end of the last rib was found and traced back to its articulation with the last dorsal vertebra, and from this point it was easy to count the spinous processes downward.

The left thumb was pressed in the space between the spinous processes, and the needle was entered about one centimetre to the right of the median line on a level with the thumb and thrust somewhat inwards and slightly upwards.

The pain appeared to be slight and the depth of the puncture varied from two to three centimetres, depending upon the age and development of the patient.

As a rule when the needle enters the canal the fluid flows, usually by drops. A little practice enables one to judge quite accurately whether the needle is in the canal or not. It is well before performing the operation for the first time to examine the p inal column of a skeleton. If the needle does not enter the

canal, or if it feels as if the point were not free, it is advisable to withdraw the needle very carefully for a short distance and not to make lateral movements with it, because this procedure is very apt to cause a slight hemorrhage, and the fluid which is obtained later is cloudy from the admixture of blood. This obscures the macroscopic, and to some extent the microscopic, appearances of the fluid.

It is well to always sterilize the needle with a wire in it so as to be sure the lumen is clear. In one case this was not done and the needle had to be withdrawn and cleaned. Raising the shoulders of the patient does not always make the fluid flow faster.

For diagnosis a small quantity of fluid is sufficient. Ordinarily four or five cubic centimetres suffices. The fluid should be collected in an absolutely clean and sterile test-tube which has previously been stoppered with cotton.

The first control experiment was made on a case of empyema. Preparations were made for an emergency, and nothing alarming occurred. The momentary pain of the puncture caused the patient to shrink and cry out, and that was all. A syringe was then applied to the needle and eight cubic centimetres of fluid were aspirated, but no ill effects were perceived.

I have performed the operation forty-five times and have never seen any ill effects, so that I feel assured it is a harmless procedure. It is obvious that large numbers are required to base accurate statistics upon, but the entire absence of any symptoms in the cases thus far observed have reassured me.

The next point which I wish to emphasize is one which has received little or no attention in the literature on the subject, and yet is of the utmost importance.

During the course of the experiments it was observed that the fluid from normal cases differed in appearance from the fluid from cases in which some inflammatory process was present in the meninges. In the former the fluid was absolutely clear, looking exactly like distilled water; while in the latter the fluid was invariably cloudy. The cloudiness was caused by a finely divided sediment suspended in the fluid. This sediment was found, on microscopic examination, to consist of mononuclear and polynuclear cells. After standing a few hours it contained more or less fibrin, evidently then an inflammatory exudation.

The normal fluid, even after standing for several days in a

sterile test-tube, showed no cloudiness or any sediment at the bottom.

Writers hitherto have laid especial stress upon finding tubercle bacilli or a more or less purulent or turbid fluid.

The finding of tubercle bacilli requires more technical knowledge of staining methods than the majority of practitioners possess, and in addition the recognition of tubercle bacilli is not invariably easy. On the other hand, to distinguish between a perfectly clear fluid like distilled water and one in which there is even the slightest cloudiness is not difficult, if the fluid is held against the light and gently shaken. Later the slight sediment which has collected at the bottom of the test-tube can be easily examined to see if it contains cells and fibrin. This gives one the diagnosis of an inflammatory exudation, and the variety of inflammation, as regards the ætiology, may be determined by appropriate methods of examination.

The importance of this seems to have been overlooked in the articles which have thus far been written on lumbar puncture. I will quote from Dr. G. W. Jacoby's article on Lumbar Puncture in the *New York Medical Journal* of December 28 and January 4, 1896, in which he refers several times to the perfectly clear fluid which may be withdrawn in tubercular meningitis.

For example, on page 8, January 4th, referring to the examination for tubercle bacilli, he says: "The best way is to allow the fluid, which is perfectly clear, as is the normal fluid, to stand in a funnel-shaped glass for twelve hours, and then to gently lift out the web-like coagulum which has formed, etc." This "web-like coagulum" is composed of fibrin, which forms later in the fluid after withdrawal, but the slight cloudiness to which I have alluded is due to cells, and is present from the first, and the fluid is not perfectly clear like the normal fluid.

Again in reference to the differential diagnosis between tubercular meningitis, purulent meningitis and abscess he says: "If a fluid is obtained which is clear and serous and contains no tubercle bacilli we may nevertheless be dealing with a case of tubercular meningitis, or even with purulent meningitis, but if, as stated, the fluid is clear and serous and we find neither pus, nor tubercle bacilli, it may nevertheless be one of those mentioned forms of meningitis, or it may be a tumor, abscess or a simple meningitis." This he says is acknowledged by all writers upon the subject and emphasized by Stadelmann recently. My

experiments, thus far, are entirely at variance with these assertions and I can only suppose that the slight cloudiness has been overlooked. Unless carefully examined the fluid often appears perfectly clear.

Any one who has searched for tubercle bacilli knows how difficult they are to find oftentimes and especially so in the cerebro-spinal fluid. In the sputum in cases of tuberculosis of the lungs where there is usually more or less destruction of the lung tissue, tubercle bacilli are not infrequently difficult to find. How much more so, then, in tubercular meningitis, where the process consists oftentimes of a few miliary tubercles in the pia at the base of the brain and along the fissures of Sylvius and Rolando, together with more or less inflammatory exudation.

In these experiments, guinea-pigs were inoculated with the fluid from those cases of meningitis upon which it seemed probable that an autopsy would not be permitted, in order to avoid the risk of losing the necessary confirmation of the diagnosis, which might have been lacking if the diagnosis had depended upon the demonstration of tubercle bacilli. I admit that I did not examine twenty, or more, cover-glass preparations as most of the observers have done. The experiments were not made with the view of demonstrating how frequently tubercle bacilli occurred in the spinal fluid, but were made to prove that the occurrence of cloudiness, however slight, due to cells, denoted an inflammatory process in the meninges and was invariably present in cases of meningitis. All inflammatory processes are accompanied by the exudation of serum and cells and there is no anatomical reason why this exudation in cases of meningitis should not become mixed with the cerebro-spinal fluid and appear in the fluid which one withdraws from the subarachnoid space, except possibly in cases of abscess of the brain it is sufficiently encapsulated to prevent this admixture. On this point I am uncertain because I have seen no cases.

Cultures were frequently taken in the normal cases as well as in the abnormal ones, simply to make the examination more complete. They were invariably sterile except in two cases of cerebro-spinal meningitis in which pure cultures of diplococcus lanceolatus were found, in one case of purulent meningitis, due to extension from the middle ear, which communicated with the air and in which a mixture of organisms was found and in a case of general infection with the staphylococcus pyogenes aureus.

The routine method of examination was as follows:

The fluid was immediately examined by holding the test-tube toward the light and gently shaking it to determine if the fluid were cloudy or not. This was done because at times the sediment is so finely divided and so slight that it might escape detection if not examined carefully. (One can have a second test-tube containing clear fluid like distilled water for comparison, but it is not essential.)

Cultures were immediately made on blood-serum in all cases in which the fluid was cloudy and in a number of normal cases. The fluid was then allowed to stand for several hours, after re-stoppering the test-tube with cotton.

If the fluid were clear at first, it remained so for days, and showed no sediment at the bottom, if taken in a sterile test-tube and not contaminated. If it were cloudy, the suspended particles settled to the bottom and formed more or less sediment, depending on the cloudiness. Usually fibrin was formed later which contained the cells in its meshes. In either case the supernatant fluid was left perfectly clear.

Cover-glass preparations were made from the sediment, dried and stained with Löffler's methylene blue, which stains the nuclei of the white corpuscles and also any bacteria which may be present. These cover-glass preparations were examined with an oil-immersion lens, although a dry lens will serve all purposes, so far as distinguishing the white corpuscles are concerned. Two or three times cover-glass preparations were stained for tubercle bacilli, but this as a rule was not done because inoculation experiments were relied upon to determine the presence of tubercle bacilli, and the cultures and Löffler's stain determined the presence or absence of pyogenic organisms.

Tests for albumin were made later by means of the nitric acid and acetic acid and heat tests. The albumin was frequently quantitated by means of the ferrocyanide of potassium and acetic acid test and a centrifugal machine. The tests for albumin were not made for its diagnostic value, but because there appeared to be an error on the part of some observers who claimed that it varied from one-half to one per cent. in the normal fluid, and abnormally often showed one to two per cent. In none of the cases thus far examined have I found more than one-tenth of one per cent. The normal fluid almost always showed a faint trace, varying from one-fiftieth to one-sixtieth of one per cent., or even less.

Tests for sugar were performed several times, and a reaction was obtained with Fehling's solution in one case. Sugar when present has no diagnostic value that I am aware of, and is present in very small quantities.

No case was examined in which the fluid was cloudy, *at the time it was withdrawn*, that was not a case of meningitis, excepting the cloudiness due to blood which occurred at times and which has been referred to, and in one case of general infection with the staphylococcus aureus, in which there was very slight cloudiness caused by the bacteria and their products, and in which there were no cells or fibrin. In those cases in which there was cloudiness caused by blood, the latter settled to the bottom in a small drop, after several hours, leaving the fluid perfectly clear and without any formation of fibrin in it.

On the other hand, in only one case of meningitis was the fluid clear when withdrawn. This case will be described later, and does not invalidate the diagnostic value of the cloudiness in the least, because in any doubtful case of diagnosis, with any methods of examination, it is necessary to repeat an experiment if it is not positive the first time; and the second puncture of this case two days later showed a cloudy fluid.

In my opinion the degree of force with which the fluid is expelled through the needle has little diagnostic value as indicating an increased amount of fluid. I have seen the fluid spurt in a fine stream in several cases in which there were no brain lesions; and, on the other hand, it has dropped from the needle in most of the cases of meningitis and in one case of hydrocephalus. In this latter case when it was punctured a second time the fluid spurted at first.

The degree of cloudiness is to some extent proportionate to the amount of cellular exudation in the meninges. In one case of purulent meningitis, caused by disease of the middle ear, the fluid was very cloudy, and a thick sediment settled quickly to the bottom of the tube. In a case of cerebro-spinal meningitis, in which the pus was confined beneath the pia, the fluid was more cloudy than in the cases of tubercular meningitis thus far examined, in which the autopsies showed that the exudation was slight and more fibrinous in character.

The microscopic examination of the sediment in the cases of meningitis thus far examined has shown a decided difference in the character of the cells which were present in the exudation.

This difference appears to be an additional factor to assist in making a differential diagnosis between tubercular meningitis and purulent meningitis. In the cases of tubercular meningitis the cells have been chiefly small round cells with a single nucleus and very little protoplasm, similar to the lymphocytes found in the blood (lymphoid cells). In addition to these there were comparatively few polynuclear leucocytes. In purulent meningitis the polynuclear leucocytes were very numerous, and the small round cells were comparatively few in number.

It is possible in the beginning of a tubercular meningitis to obtain a perfectly clear fluid. This was exemplified in one case in which two subsequent punctures showed the fluid to be slightly cloudy and to contain small round cells and fibrin. At the autopsy of this case only a few miliary tubercles were found at the vertex of the brain and but slight inflammatory exudation, also at the vertex.

Subsequent cloudiness of a clear fluid occurred in two cases after the fluid had remained two or three days in the test-tubes. Cultures and cover-glass preparations showed this to have been caused by bacteria, and there were no cells present. Evidently the organisms had been introduced in examining the fluid after its withdrawal from the spinal canal.

In a certain number of cases numerous white particles were present in the fluid when it was withdrawn. They were neither cells nor fibrin, and showed a tendency to dissolve after several hours. Solutions of corrosive sublimate and alcohol were added to normal fluid without causing the particles to appear. The skin was moistened and scraped and the scrapings examined, also with a negative result. These particles appeared in the fluid withdrawn from cases in which there was no meningitis. They should not be confounded with the cloudiness due to cells. The latter is very finely divided and gives a general haziness to the fluid. The particles may occur in cases of meningitis, but they do not interfere with the detection of the general cloudiness.

In the diagnosis of hemorrhage into the brain or subarachnoid space I have had no experience, but several times a number of drops of blood have followed the puncture, soon changing to blood-tinged fluid. In these cases a second puncture has evacuated clear fluid, so that for diagnostic purposes I should think that a considerable quantity of blood would be necessary. In Dr. G. W. Jacoby's two cases I believe the blood had undergone changes and was dark-colored.

The results of lumbar puncture have thus far shown it to have no therapeutic value. Some observers have noted a temporary relief of symptoms following the operation. Washing out the subdural space, which has recently been suggested by Dr. G. W. Jacoby, seems to me to be impractical as a curative measure, except possibly in those cases of meningitis due to extension from neighboring parts. In cases of cerebro-spinal meningitis the exudation is chiefly beneath the pia and cannot be removed by washing; and in tubercular meningitis the tubercles themselves cannot be removed. Furthermore tubercular meningitis is almost invariably the end process of a more or less extensive general tuberculosis, and for this reason can hardly be compared with those cases of tubercular peritonitis in which the process may be a local one, and which have been relieved or cured (?) by laparotomy.

In the description of the cases which follow I have omitted, for the sake of brevity, most of the clinical details because they had no bearing on the results of the investigation. The same applies to the autopsy reports.

Case I.—Female; aged 29 months. Entered hospital December 8, 1895. Died December 19, 1895. Diagnosis, tubercular meningitis.

Clinical History.—Vomiting two weeks previously; irritability; fever; attacks of screaming.

Physical Examination.—Emaciated; anæmic; irritable when roused; apathetic; retraction of head; moderately elevated temperature until day of death when it rose to 43° C. (109.8° F.) Progressive symptoms; stupor; convulsions.

Puncture performed at time of death.—About 4 c.c., of turbid fluid were withdrawn. The fluid contained about $\frac{1}{30}$ of one per cent. of albumin. Microscopic examination showed the presence of numerous small mononuclear cells, a few polynuclear leucocytes and fibrin. Two cover-glass preparations were stained for tubercle bacilli and none were found. An autopsy was not obtained. This case occurred before the systematic examinations were begun and a guinea-pig was not inoculated.

Case II.—Female; aged 20 months. Under observation for ten weeks. Diagnosis, empyema, chronic interstitial pneumonia and cerebro-spinal meningitis.

First Puncture January 16, 1896.—8 c. c., of perfectly clear, colorless fluid withdrawn by aspiration. No symptoms attended or followed the operation. The fluid contained a faint trace of albumin (about $\frac{1}{50}$ of one per cent.). The fluid remained clear for several days and showed no sediment at the bottom of the test-tube.

Second Puncture January 23d.—About 7 c. c., of clear fluid were withdrawn, similar in all respects to the first. No reaction on the part of the patient.

Third Puncture February 16th.—This was performed on the day of the patient's death. There was some retraction of the head for a day and a half preceding the patient's death; considerable apathy and a slight elevation of temperature. A somewhat cloudy fluid was withdrawn which contained $\frac{1}{10}$ of one per cent. of albumin. Microscopic examination of the sediment showed chiefly polynuclear leucocytes and fibrin. Cover-glass preparations stained with carbol-fuchsin showed numerous lancet-shaped diplococci surrounded by capsules. (Diplococcus lanceolatus.)

Autopsy Report.—The vessels of the pia much injected. Purulent exudation under the pia, distributed over the vertex and base of the brain and extending down on the cord. The pus was chiefly confined to the lymph-spaces by the sides of the vessels. Cultures on blood-serum showed a pure culture of the diplococcus lanceolatus. The remaining organs showed a right-sided empyema with perforation into the lung; chronic interstitial pneumonia; no evidence of tuberculosis in any organs.

Case III.—Female; aged 4 months. Under observation for seven weeks. Diagnosis, infantile atrophy.

Puncture January 17, 1896.—5 c. c., of perfectly clear fluid were withdrawn. No symptoms attended or followed the operation. The patient died on January 22d.

Autopsy Report.—Brain normal. The remaining organs showed the changes common to infantile atrophy, *viz.*, areas of atelectasis in the lungs; slight hyperplasia of the mesenteric lymph glands; anæmia of the organs.

Case IV.—Female; aged 10 months. Under observation for six weeks. Diagnosis, infantile atrophy.

Puncture January 23, 1896.—5 c. c., of clear, colorless fluid were withdrawn, which contained a faint trace of albumin, (about $\frac{1}{60}$ of one per cent.). The fluid remained clear and without sediment at the bottom of the test-tube. Patient discharged relieved on February 24th.

Case V.—Male; aged 3½ years. Under observation for five days. Diagnosis, primary tuberculosis of the intestines; pneumonia and icterus.

Clinical History.—The temperature was normal when he entered the hospital on January 31st, but on the third day it rose to 40° C. (104° F.). From this time until his death, a day and a half later, there were constant tonic spasms and trismus. The pulse and respiration were but slightly accelerated. There was marked stupor.

Puncture February 3, 1896.—About 5 c. c., of clear, colorless fluid were withdrawn. There was no sediment at the bottom

of the test-tube after standing. Cultures made from the fluid were sterile. The patient died on February 4th.

Autopsy Report. (Dr. F. B. Mallory).—Brain normal. Primary tuberculosis of the intestines; double pneumonia.

Case VI.—Male; aged 6 months. Seen only once on February 1st. Diagnosis, tubercular meningitis.

Clinical History.—Vomiting; feverishness and retraction of the head, which began about the middle of January. There was some abatement of the symptoms after several days. On January 27th, the vomiting recommenced, together with retraction of the head; apathy and twitching of the left arm and leg. The temperature on February 1st was 38.4° C., (100° F.).

Puncture February 1, 1896.—About 2 c. c., of cloudy fluid were withdrawn. No reaction on the part of the patient attended the operation. Microscopic examination showed the cloudiness to be chiefly due to small mononuclear cells. Some fibrin was formed later. Cover-glass preparations were stained for tubercle bacilli and two doubtful ones were found. Cultures on blood-serum were sterile. A guinea-pig was inoculated with the fluid but he was unfortunately killed by mistake and not examined.

Upon inquiry, it was learned that the child died three weeks later. During the last three days she had opisthotonos and convulsions.

Case VII.—Male; aged 7 months. Under observation for three weeks. Diagnosis, porencephalus.

Clinical History.—The patient entered the hospital on February 5, 1896. There was a history of frequent convulsions which began when he was about three months old. While in the hospital the convulsions occurred not less than twenty times a day. Oftentimes he had several in an hour. Marked rigidity of the extremities and opisthotonos were constant symptoms. The temperature was normal until ten days before his death. Physical examinations of the various organs, made several times, were invariably negative. Seven or eight days before his death he developed an acute inflammation of both middle ears, which subsided in two or three days without any discharge.

First Puncture, February 5, 1896.—12 c. c. of clear, colorless fluid were withdrawn. The fluid escaped at first with a spurt, and then by drops. The specific gravity was 1006, and there was no albumin by the heat and nitric acid tests. A test for sugar with Fehling's solution was negative. No sediment collected in the test-tube after standing.

Second Puncture, February 21st.—A clear fluid was withdrawn, similar in all respects to the first except that it contained a faint trace of albumin (about $\frac{1}{8\,0}$ of one per cent.). This time the fluid escaped by drops.

Third Puncture, February 27th.—The fluid was perfectly

clear and dropped slowly from the needle. It remained clear and contained a faint trace of albumin.

After standing for ten days, the fluid from the second puncture became cloudy. Microscopic examination showed that the cloudiness was due to bacteria. There were neither cells nor fibrin present.

Autopsy Report.—The brain showed a rudimentary development, but no evidences of inflammation. The other organs were normal, with the exception of areas of atelectasis in the lungs. Cultures from the organs showed the colon-bacillus present in the liver, spleen and kidney and a mixture of organisms in the lungs.

Case VIII.—Male; aged 5 years. Under observation for three weeks. Diagnosis, tubercular meningitis; disseminated tuberculosis of the lungs, liver, spleen and kidneys; chronic tuberculosis of the bronchial glands.

Clinical History.—There was an indefinite history of slight convulsions, without loss of consciousness, which occurred just before he entered the hospital, together with headache for a week and vomiting. The patient came to my out-patient clinic February 20, 1896, and was recommended for admission into the hospital. While undergoing examination he became quite rigid. This was immediately followed by spasmodic movements of the right leg and arm and athetoid movements of the fingers of the right hand. There was no loss of consciousness. The child seemed frightened and clung to his mother's neck. After his admission to the hospital he had two or three similar attacks. About a week later (February 27th), he showed the classical symptoms of tubercular meningitis: vomiting, headache, apathy, slow and intermittent pulse, slight temperature, tâche cerébral, somewhat retracted abdomen.

First Puncture March 3d.—5 c. c. of perfectly clear fluid were withdrawn. At first the fluid spurted and then flowed by drops. Cultures taken from the fluid when it was withdrawn were sterile. Three days later the fluid was cloudy and cultures showed the staphylococcus aureus. There were neither cells nor fibrin present.

Second Puncture March 5th.—9 c. c. of very slightly cloudy fluid were withdrawn. The cloudiness was caused by a finely divided sediment suspended in the fluid, and was difficult to detect until the test-tube was held toward the light and gently shaken. Microscopic examination of the sediment, after standing, showed the presence of a few polynuclear leucocytes, many round cells with a single nucleus, and fibrin. Löffler's methylene blue solution was used to differentiate the cells. No microörganisms were found, and cultures from the fluid were sterile. The fluid contained a faint trace of albumin (less than $\frac{1}{2}\frac{1}{5}$ of one per cent.).

Up to this time there had been considerable uncertainty about the diagnosis. The possibility of a cerebral tumor could not be denied, and there were a number of symptoms in the case which pointed strongly toward it. After the second puncture the diagnosis of tubercular meningitis was made. The patient was operated upon by Dr. H. L. Burrell on March 6th.

Third Puncture March 9th.—About 10 c. c. of slightly cloudy fluid were withdrawn, similar in all respects to that which was obtained from the second puncture. A guinea-pig was inoculated with some of this fluid and developed tuberculosis. The patient died on March 12th.

Autopsy Report (Dr. F. B. Mallory).—"On the pia, on both sides, at the vertex, centering over the fissures of Rolando and Sylvius, and dipping down in the longitudinal fissure were two areas about 6 c. long and 4 c. wide, dotted with yellowish specks the size of pin-heads. These specks were more numerous on the right side than on the left. Close to and in the longitudinal fissure they fused together forming yellow areas which resembled fibrino-purulent exudation. No tubercles were found at the base of the brain or in the fissure of Sylvius. The inner surface of the dura on the right side showed a number of single and agglomerated tubercles." The remainder of the report is omitted.

Autopsy Diagnosis.—Chronic tubercular bronchial glands; disseminated tuberculosis of the lungs, spleen, kidneys and liver, miliary tubercular meningitis.

It is not surprising to me that the fluid from the first puncture was clear, but rather that the fluid was cloudy from the two subsequent punctures, considering the small quantity of inflammatory exudation and its location.

Case IX.—Male; 5 years of age. Under observation for two weeks after the first puncture. Diagnosis, tuberculosis of the hip joint; glandular tuberculosis; disseminated tuberculosis of the lungs, liver, kidneys and spleen; tubercular meningitis.

Clinical History.—The patient was operated upon for a hip abscess, and a day or two afterward developed symptoms of meningitis. There was vomiting, irritability and apathy in the beginning, and later, stupor and convulsions supervened.

First Puncture March 2d.—About two or three days after the operation several cubic centimetres of slightly cloudy fluid were withdrawn. After standing several hours there was a web-like coagulum of fibrin which extended up from the bottom of the test-tube for some distance. Microscopic examination showed numerous mononuclear cells, and some polynuclear leucocytes, together with considerable fibrin. Culture taken on blood-serum were sterile. The fluid contained a faint trace of albumin, (about $\frac{1}{30}$ of one per cent.).

Second Puncture March 5th.—21 c. c., of slightly cloudy fluid were withdrawn. The first 14 c. c., escaped quite rapidly but

after that the fluid dropped quite slowly. The fluid and sediment were similar to that obtained from the first puncture. A test for sugar with Fehling's solution gave a negative result.

Third Puncture March 9th.—12 c. c., of fluid were withdrawn which was more cloudy than the fluid from the two previous punctures. In other respects the fluid and sediment did not differ from that obtained before. A guinea-pig was inoculated with some of the fluid and developed tuberculosis.

Fourth Puncture March 12th.—Several cubic centimetres of cloudy fluid were withdrawn which was similar in all respects to that which was obtained from the previous punctures. The patient died on March 16th.

Autopsy Report.—The vessels of the pia were injected and there was considerable fibrinous exudation in the meshes of the pia, especially at the base of the brain and along the fissures of Sylvius and Rolando. The ventricles contained more fluid than normal and the surface of the ependyma was covered with fine pin-point elevations. The lungs, liver, spleen and kidneys showed a disseminated tuberculosis and the bronchial and mesenteric lymph glands were enlarged and contained numerous "cheesy" foci.

Case X.—Premature infant of 8 months. Under observation for ten days.

Clinical History.—The day before its death the temperature rose to 41° C. (105.8° F.). No cause could be detected by physical examination.

Puncture March 5th (the day of its death).—2 c. c., of blood-tinged fluid were withdrawn. The operation was unattended by symptoms. Microscopically, the fluid contained red blood corpuscles and a few white corpuscles, but no fibrin. There was a faint trace of albumin in the fluid (about $\frac{1}{60}$ of one per cent.).

Autopsy Report.—The lateral ventricles were dilated and contained an excess of fluid and macerated brain substance. Cultures taken from this fluid were sterile. The meninges showed no evidences of inflammation. The other organs were not abnormal with the exception of areas of atelectasis in the lungs. Cultures from the organs showed the presence of the colon bacillus.

Case XI.—Male; aged 4 months. Still under observation. Diagnosis, infantile atrophy.

Physical Examination.—Emaciation; pallor and lack of development, otherwise negative. The temperature was slightly subnormal.

Puncture on March 8th.—The fluid was at first slightly tinged with blood but later it was perfectly clear and showed no sediment after standing. No symptoms attended or followed

the operation. The fluid contained a faint trace of albumin, (less than $\frac{1}{50}$ of one per cent.).

Case XII.—Male; aged 4 months. Under observation for seventeen days. Diagnosis, rhachitis and atrophy.

Puncture on March 8th.—3 c. c. of clear fluid were withdrawn, which contained a faint trace of albumin (less than $\frac{1}{50}$ of one per cent.). The fluid showed no sediment after standing for several days. The operation had no effect on the patient.

He was discharged, relieved, on March 13th.

Case XIII.—Female; aged 1 month. Under observation for nineteen days. Diagnosis, obstetrical paralysis of left arm.

Puncture on March 8th.—2 c. c. of slightly blood-tinged fluid were withdrawn. Microscopic examination showed the presence of red blood corpuscles and an occasional white corpuscle, but no fibrin. The operation was without effect on the patient. Discharged well on March 14th.

Case XIV.—Male; aged 2 years. Diagnosis, acute purulent meningitis.

Clinical History.—The patient had a chronic purulent inflammation of the right ear and was operated on by Dr. H. L. Morse on March 6, 1896. The patient died on March 10th, with symptoms of meningitis.

Puncture made six and a half hours after death.—A very turbid fluid was withdrawn, and a purulent, yellow sediment settled quickly to the bottom of the test-tube. Cover-glass preparations of the sediment were stained with Löffler's methylene blue solution, and showed the presence of numerous polynuclear leucocytes, fibrin and bacteria.

Autopsy Report.—Both cerebral hemispheres, between the pia and the dura, were covered with thick, foul-smelling pus. The exudation extended to a lesser degree over the base of the brain. There were three openings on the upper surface of the right temporal bone through which a probe could be passed through the middle ear into the external meatus. Corresponding to these openings, there was a small abscess in the right temporo-sphenoidal lobe of the brain. Cultures made from the pus showed a mixture of organisms.

Case XV.—Male; aged 17 months. Still under observation. Diagnosis, rhachitis, with spasm of the larynx and convulsions.

Clinical History.—The patient entered the hospital on March 9, 1896, with a history of convulsions, at intervals, for the last two months. Aside from marked evidences of rhachitis, the physical examination was negative. The infant was fat and looked perfectly healthy. The temperature was normal at the time of entrance, and remained so. During the first two weeks he had a number of convulsions, which were always preceded by spasm of the larynx.

Puncture on March 11th.—Several cubic centimetres of blood-tinged fluid were withdrawn. After standing, the blood collected into a small drop at the bottom of the test-tube and left the fluid perfectly clear. Microscopic examination of this drop showed it to consist of red blood corpuscles, together with a corresponding number of white corpuscles interspersed. There was no fibrin. The fluid contained a faint trace of albumin. There were no symptoms attending or following the operation.

Case XVI.—Male; aged 21 months. Under observation for sixteen days. Diagnosis, rhachitis, bronchitis and broncho-pneumonia. Patient entered hospital March 5th.

Puncture on March 11th.—3 c. c. of clear, colorless fluid were withdrawn, which contained a faint trace of albumin (about $\frac{1}{50}$ of one per cent.). No sediment collected in the test-tube after standing. The operation was without effect on the patient.

Later History.—The patient was discharged well of bronchitis and broncho-pneumonia.

Case XVII.—Male; aged 7 months. Under observation for two weeks. Diagnosis, pulmonary, glandular and disseminated tuberculosis.

Physical Examination.—Extreme emaciation and evidences of consolidation in both lungs. Temperature slightly elevated.

Puncture on March 11th.—3 c. c. of perfectly clear fluid were withdrawn, which contained a faint trace of albumin (about $\frac{1}{60}$ of one per cent.). The fluid remained without sediment for several days. No symptoms attended or followed the operation. The patient died on March 16th.

Autopsy Report.—There was very extensive tuberculosis of both lungs and considerable destruction of the lung tissue in the upper and lower lobes of the right lung. Acute miliary tuberculosis of the liver, spleen and kidneys and "cheesy" (tuberculous) bronchial and mesenteric lymph glands. The brain was carefully examined for evidences of tubercular meningitis, but neither tubercles nor inflammatory exudation were found. The ventricles were normal.

Case XVIII.—Female; aged 4 years. Entered hospital on March 11th, and died on March 12, 1896. Diagnosis, chronic tuberculosis of the dorsal vertebræ and bronchial lymph glands; tubercular meningitis; acute miliary tuberculosis of the lungs, spleen, liver and kidneys.

Puncture (three hours after death).—A cloudy fluid was withdrawn. Microscopic examination of the sediment showed numerous small mononuclear cells, a few polynuclear leucocytes and fibrin. Cultures were sterile.

Autopsy Report (Dr. F. B. Mallory).—"The convolutions were much flattened. There were numerous miliary tubercles in the pia over the convolutions, but the largest number were found in

the fissure of Sylvius and at the base of the brain. Tubercles were also found in the gray and white matter of the occipital and frontal regions and also in the white matter of the cerebellum. The ependyma was studded with minute pin-point elevations." (The remainder of the report is omitted.)

Case XIX.—Female; aged 6 months. Under observation for thirteen days. Diagnosis, slight rachitis and anæmia; persistent vomiting.

Clinical History.—The patient was sent to the hospital with a provisional diagnosis of tubercular meningitis. There was a history of irritability, obstinate vomiting and frequent attacks of crying. The physical examination made on March 16th, two days after coming to the hospital, was negative except that the infant was pale and looked ill. The temperature was practically normal during her stay in the hospital.

Puncture on March 16, 1896.—Several cubic centimetres of perfectly clear fluid were withdrawn. The fluid contained no sediment after standing for several days. The patient was not affected by the operation.

The child was fed upon modified milk, once or twice by means of a stomach-tube, and after several days ceased to vomit and took her food quite well. It was afterward learned that the infant had been given coffee for some time. She was discharged relieved.

Case XX.—Male; aged 11 months. Under observation for about six weeks. Diagnosis, rhachitis, anæmia and atrophy.

Clinical History.—During his stay in the hospital, the patient gained somewhat in weight and was otherwise improved. The physical examination showed marked evidences of rhachitis consisting of deformity of the thorax, epiphyseal enlargement, a well marked "rosary," etc. In addition the patient was very much emaciated and anæmic. The examination of the organs was negative.

Puncture on March 18th.—Several cubic centimetres of clear fluid were withdrawn without producing any effect on the patient. The fluid contained no sediment after standing. The patient was discharged relieved, about three weeks later.

Case XXI.—Male; aged 7 months. Under observation for one week. Diagnosis, rhachitis and atrophy.

Clinical History.—The patient showed evidences of rhachitis and was much emaciated. The temperature was normal.

Puncture on March 18th.—Several cubic centimetres of clear fluid were withdrawn without producing any effect on the patient. There was no sediment in the fluid after standing for several days.

The patient was discharged, after remaining a week in the hospital, relieved of some indigestion which he had at entrance.

Case XXII.—Male; aged 7 months. Under observation about twelve days. Diagnosis, hydrocephalus.

Clinical History.—The patient entered the hospital on March 21st for the purpose of having some of the fluid withdrawn from the brain by means of lumbar puncture. Measurements were as follows:

Head, circumference,	55	cm.
Antero-posteriorly,	38	"
Across the vertex of the head,	33	"
Thorax, at level of nipple, circumference,	37½	"
Abdomen, at level of umbilicus, circumference,	39	"
Length of child,	67½	"

First Puncture on March 22d.—105 c. c. of clear fluid were withdrawn. At times, when the infant moved, the fluid contained blood, but became clear again. The withdrawal of the fluid did not appear to affect the patient unpleasantly. The pulse was a little slower temporarily. The withdrawal of the fluid caused the fontanelle and sutures, which were widely open and tense, to become soft and concave. The fluid contained no sediment after standing for several days. The fluid contained a faint trace of albumin ($\frac{1}{40}$ of one per cent.).

Second Puncture on April 1st.—The patient had been vomiting for two or three days previous to the second puncture, and was rather somnolent; 5 c. c. of perfectly clear fluid were withdrawn, which contained no sediment after standing for several days.

The ventricles were washed out a day or two later by Dr. Augustus Thorndike, and the child died the same day. No autopsy was obtained.

Case XXIII.—Male; aged 3½ months. Under observation for two weeks. Diagnosis, pulmonary tuberculosis, chronic tuberculosis of the bronchial and mesenteric lymph glands, disseminated tuberculosis of the liver, spleen and kidneys.

Clinical History.—The infant was said to have had pneumonia for a week when he entered the hospital March 13, 1896. During his stay in the hospital the temperature ranged from 36.4° C. (97.5° F.) to 38.5° C. (101.3° F.). The physical examination showed extreme emaciation and evidences of consolidation in both lungs. For five or six days preceding his death there was considerable apathy and some retraction of the head.

Puncture on March 24th.—Several cubic centimetres of perfectly clear fluid were withdrawn, which contained no sediment after standing for several days. The operation produced no effect upon the patient. The infant died on March 27th.

Autopsy Report.—There was extensive tuberculosis of both lungs, with some destruction of the lung tissue in the left upper lobe; extensive chronic ("cheesy") tuberculosis of the bronchial and mesenteric lymph glands; miliary tuberculosis of the liver, spleen and kidneys.

With the exception of injection of the vessels of the pia the brain was normal. Careful search was made for miliary tubercles and exudation in the meninges with negative result. The ependyma was perfectly smooth.

Case XXIV.—Male; aged 10 months. Under observation for four days. Diagnosis, tubercular meningitis.

Clinical History.—The patient entered the hospital April 9, 1896. There was a history of three weeks' illness, which began with vomiting. The infant was irritable and restless, and during the last four days he was apathetic. The physical examination was negative, with the exception of considerable apathy. The temperature was normal while in the hospital.

First Puncture on April 9th.—5 c. c. of very slightly cloudy fluid were withdrawn. It was necessary to compare the fluid with some perfectly clear fluid in order to be certain of the cloudiness. On the following day the fluid contained a scarcely perceptible web of fibrin. Microscopic examination showed a number of small mononuclear cells, a few polynuclear leucocytes and fibrin. Cultures were sterile.

Second Puncture on April 11th.—About 10 c. c. of fluid were withdrawn, which was more cloudy than the fluid from the first puncture. Microscopic examination of the sediment showed numerous small mononuclear cells, a few polynuclear leucocytes and fibrin. Cultures were sterile. A guinea-pig was inoculated with 2½ c. c. of the fluid and developed tuberculosis.

Upon inquiry it was learned that the infant died on April 21st. There was a history of convulsions during the last three days.

Case XXV.—Female; aged 5 years. Still under observation, Diagnosis, probable cerebral tumor.

Clinical History.—There was a history of vomiting, headaches and dizziness, which had occurred more or less frequently for six months. The patient was rather stupid when she entered the hospital on April 14, 1896, but this symptom disappeared after a day or two and has not recurred. An examination of the eyes showed optic neuritis, probably in a receding stage. The ears were normal. The patient has had a number of attacks of headache, and the afternoon temperature has been slightly elevated most of the time. The urine was negative.

First Puncture on April 16th.—Several cubic centimetres of clear fluid were withdrawn without causing any effect on the patient. There was no sediment in the fluid after standing. Cultures were sterile. The fluid contained a faint trace of albumin (less than $\frac{1}{50}$ of one per cent.). Equal parts of the fluid and Fehling's solution, showed a reddish tinge and a very slight precipitation.

Second Puncture April 28th.—About 15 c. c. of fluid were withdrawn, which was tinged with blood at first and afterwards

became clear. The patient cried while the puncture was being made and continued to cry afterward, complaining of headache. The fluid contained no sediment after standing. Cultures were sterile. The fluid contained no albumin by the heat and acetic acid test.

Case XXVI.—Female; aged 4 years. Under observation for two days. Diagnosis, acute suppurative myositis; acute purulent peri-carditis; acute fibrinous pleurisy; multiple abscesses of heart, lungs, kidneys, liver and brain.

Clinical History.—The patient entered the hospital on April 27, 1896. She was said to have been well until one week before, when she complained that her hip was "sore." Swelling of the right thigh was noticed on April 26th. Up to this time she had played about the house. On April 27th, a loud pericardial friction rub was heard, which disappeared on the next day. The patient showed evidences of severe infection consisting of somnolence, high temperature, rapid pulse and respiration and cyanosis. The patient died on April 28th.

Puncture April 28th.—5 c. c., of very slightly cloudy fluid were withdrawn. On the following day the cloudiness persisted and there was no sediment at the bottom of the test-tube. Microscopic examination failed to detect any cells, or fibrin. A relatively small number of micrococci were found. The fluid contained less than $\frac{1}{4\,0}$ of one per cent. of albumin. Cultures on blood-serum showed between forty and fifty colonies of staphylococcus pyogenes aureus.

Autopsy Report (Dr. F. B. Mallory).—"Convolutions not flattened in the least. In the floor of the left ventricle, in the anterior portion of the lenticular nucleus, was a dark red area, 2 m. m., in diameter, with slightly softened centre on section. Section throughout the brain substance showed two similar, but smaller, areas lying in the gray matter of the cortex. Nothing abnormal found in the cortex, or cord." The remainder of the report is omitted.

Case XXVII.—Female; aged 3½ months. Under observation for several days. Diagnosis, infantile atrophy.

Clinical History.—The patient was sent to the hospital from my out-patient clinic because she had been somewhat stupid for a day or two and there was a question of tubercular meningitis. The temperature was slightly elevated.

Puncture on May 3d.—Several cubic centimeters of blood tinged fluid were withdrawn which contained less than $\frac{1}{5\,0}$ of one per cent. of albumin. A small drop of blood collected in the bottom of the test-tube after standing, and left the fluid clear. No formation of fibrin occurred.

After two days, the infant was as bright as usual and was sent home.

Case XXVIII.—Male; aged 2 years and 3 months. Under observation for several days. Diagnosis, hydrocephalus.

Clinical History.—The patient entered the hospital on May 9th, with the history that he had not been able to walk for three weeks. The physical examination was negative with the exception of increased patellar reflexes and a head which was larger than normal. The measurements of the head were as follows: circumference, 53.5 cm.; antero-posterior measurement, 34 cm.; transverse measurement, 30 cm.

Puncture May 11th.—About 7 c. c., of blood-tinged fluid were withdrawn, which contained $\frac{1}{60}$ of one per cent. of albumin. Cultures were sterile. A small drop of blood collected in the bottom of the test-tube after standing. There was no formation of fibrin.

Case XXIX.—Female; aged 3 years. Under observation for seven days. Diagnosis, tubercular meningitis; chronic tuberculosis of the bronchial glands; acute miliary tuberculosis of the lungs.

Clinical History.—The patient entered the hospital May 11, 1896. There was a history of measles six weeks before, followed by a mild attack of broncho-pneumonia. The present disease began a week before her entrance into the hospital, with vomiting. There was a purulent discharge from the left ear which had lasted two or three days. On May 11th, the patient was somnolent and the extremities were somewhat rigid, otherwise the physical examination was negative. The temperature was somewhat elevated.

First Puncture May 11th.—About 10 c. c., of slightly cloudy fluid were withdrawn which contained $\frac{1}{30}$ of one per cent. of albumin. A slight coagulum of fibrin formed in the fluid after standing. Microscopic examination showed numerous small mononuclear cells, a few polynuclear leucocytes and fibrin. Cultures were sterile.

Second Puncture May 12th.—Dr. S. J. Mixter trephined the skull over the left ear and tapped the lateral ventricle, but obtained no fluid. Lumbar puncture was then performed and 50 c. c., of slightly cloudy fluid were withdrawn, similar in all respects to that which was withdrawn the day before. A portion of the brain substance which was tense and congested, and which protruded through the trephine opening, gradually grew smaller and paler and sank below the edge of the skull as the fluid was withdrawn from below. After the effects of the ether had passed off, the patient was brighter than she had been during the day.

Third Puncture May 13th.—The patient's general condition was not as good. There was marked stupor, inability to swallow, the pupils were unequal and did not react to light, the pulse was rapid and the temperature was somewhat elevated. About

40 c. c. of slightly cloudy fluid were withdrawn, which was similar to that withdrawn before. After the withdrawal of the fluid the patient did not rouse as she had done on the day before. A guinea-pig was inoculated with 2 c. c. of the fluid obtained from the second puncture.

Fourth Puncture May 16th.—About 7 c. c. of slightly cloudy fluid were withdrawn, similar in all respects to that obtained before. The patient died on April 18th.

Autopsy Report (Dr. Wm. F. Whitney).—"Dura not adherent to calvaria. The meshes of the pia over the cortex filled with a slightly yellowish-green exudation. In left parietal lobe, opposite the wound, the brain substance was softened and reddened, evidently the result of the operation. Over the base of the brain, especially in the region of the pons, a thick, yellowish, fibrinous exudation was present, completely obscuring the outlines. On section of the ventricles a little opaque fluid escaped. The brain substance was pale, soft and moist. There were miliary tubercles in the meshes of the pia. A hemorrhagic line could be followed from the wound to the candate nucleus of the left side. In the exudation on the base of the brain numerous opaque granulations were found, some of them apparently caseous. There was no inflammation of the temporal bone." Remainder of report omitted. Abdomen not examined.

SUMMARY:

1. The normal cerebro-spinal fluid contains neither cells nor fibrin, and is perfectly clear.

2. In cases of meningitis the cerebro-spinal fluid is *invariably* cloudy when withdrawn. The degree of cloudiness is to some extent proportionate to the amount and character of the exudation in the meninges.

3. The cloudiness is caused by cells. The character of the cells differs with the variety of the meningitis. After withdrawal, more or less fibrin is formed in the fluid. The presence of these cells and fibrin is pathognomonic of inflammation in the meninges.

4. The cloudiness is oftentimes so slight that close observation is necessary to detect it.

5. The operation is not difficult to perform on infants and children. It is not dangerous, if strict cleanliness is observed.

6. The differential diagnosis between the various kinds of meningitis can be made by microscopic examination of the sediment, by cultures taken from the fluid and by inoculation experiments.

7. Inoculation experiments afford the surest means of deter-

mining tubercular meningitis. It is of value to distinguish between the varieties of meningitis in order to determine if tubercular meningitis is recovered from.

8. In the normal fluid, a faint trace of albumin is usually present, about $\frac{1}{500}$ of one per cent., or less, by quantitative analysis. In meningitis the amount of albumin is increased, and has varied from $\frac{1}{300}$ to $\frac{1}{100}$ of one per cent.

9. In one case a diagnosis of general infection with the staphylococcus pyogenes aureus was made from cultures taken from the cerebro-spinal fluid.

10 EXETER STREET.

A CASE OF LUMBAR PUNCTURE OF THE SUBARACHNOID SPACE.

BY CHARLES GODWIN JENNINGS, M.D.,

Detroit.

This case is of interest because of the accident that occurred from injudicious manipulation, the sharp rise of temperature following the operation, and the micro-organism found in the cerebro-spinal fluid.

The patient, aged six years, with no family history of tuberculosis, was a strong, healthy girl until the first part of January, 1896, about eight weeks before her death. At that time she began to fail in health, although she was not so ill that her parents thought it necessary to consult a physician.

She was seen March 13, by the family physician, Dr. W. B. Sprague, and by me in consultation March 21. Clinical diagnosis, tubercular meningitis.

At the time of the puncture she was comatose, with dilated, uneven and sluggish pupils, gummy conjunctival secretion, general tremor—the usual phenomena of the stage of effusion.

Lumbar puncture of the subarachnoidal space was made, and 24 c.c. of fluid withdrawn. A large needle from an antitoxin syringe was used. The puncture was not perfectly made as the fluid flowed freely only when the needle was depressed. Too much force was accidentally used in depressing the needle, and it broke deeply in the tissues of the back. A deep incision, and a search as thorough as seemed justified without a more careful antisepsis than was at that moment possible, failed to reveal the needle. Although this was the result of injudicious manipulation, the unpleasant accident should be remembered as a possibility by the operator.

A few hours after the puncture the temperature rose to $104\frac{4}{5}°$, and the pulse to 136. Up to that time the temperature had not risen above 101° or the pulse above 100. The child aroused somewhat from the coma, the pupils became more responsive, and the tremor ceased.

On the following day the puncture was repeated, and eight drams of fluid were withdrawn. After the second puncture

there was a partial return of consciousness and a general temporary relief of the pressure phenomena. The temperature curve was not disturbed by this second puncture. She died, however, two days later. There was no autopsy.

The fluid withdrawn was clear, depositing a few flocculi of fibrin after standing. Bacteriological examination by Dr. E. H. Sargent demonstrated the presence, in large numbers, of the pneumococcus. Culture experiment was not made.

457 Jefferson Avenue.

DISCUSSION ON LUMBAR PUNCTURE.

Dr. J. Henry Fruitnight.—The operation in most cases is harmless. Still, it has been reported that there have been some cases of sudden death following lumbar puncture. In a series of eighty-six cases reported, there were four deaths. Five cases of cerebellar tumor were reported, in which the operation was performed, and in three of these death occurred suddenly in the course of from six to forty hours after the operation. The sudden death was explained by the fact that there is a change in the nutrition of the organ by the withdrawal of the fluid, and in consequence there is compression of the cerebral tissues by the bony edges of the occipital bone. I have seen this operation done often without any bad results, but I think we should remember that sometimes death does follow quite suddenly.

Dr. A. D. Blackader.—We have used this in two or three cases in the Montreal General Hospital. In one case there seemed to be a very decided improvement in the symptoms for some time. In another case, although there was a considerable amount of fluid removed there was comparatively little improvement, and the child sank rapidly. It seems to me that it would depend a good deal on the site of the inflammation whether there would be a favorable result upon the symptoms. I think it may prove a guide in differential diagnosis. It would appear that it would only seldom be of value in giving relief if there is severe inflammation. We must expect communication between the cavities of the brain and the cord to be sometimes interrupted. At the same time one cannot help feeling that since medical aid is of so little value in this disease, that we must look, perhaps to surgical aid. Operative interference in tubercular peritonitis has been of such marked value occasionally in children, that we may look forward, perhaps, to some successful surgical interference in tubercular meningitis. I must say that I think that actual trephining of the skull or allowing the entrance of air to the membranes of the brain might offer some help in limiting in-

flammatory conditions, by some inhibitory or actual destructive effect upon the tubercle bacilli. We know that it has in abdominal peritonitis; why should it not have some effect in tubercular meningitis?

Dr. L. Emmett Holt.—I have only employed lumbar puncture three times, and in each instance, extracted quite a large quantity of fluid; at one time nearly three ounces were removed, and at another, over one and one-half ounces. To my great surprise there was absolutely no change in the symptoms. The child was in deep coma in the last stages of tubercular meningitis, and lived four days after the operation. Two punctures were made in this case, and no tubercle bacilli were found after careful search either in the fresh fluid or by culture. The case was a typical one. For differential diagnosis in the different forms of meningitis it seems to me not likely to prove of great value, for in Dr. Caillé's cases they were absent in two out of six cases.

Dr. Caillé.—I think there can be no doubt as to the diagnostic value of the procedure. There are very many cases on record now in which the liquid has given positive results and a positive diagnosis.

It is difficult to find tubercle bacilli, but the experts generally do find it. I admit that in two out of seven, or two out of six of my cases it was not found; it might have been found at a subsequent examination. I would say that for any one who is expert it is not necessary to use a syringe: I use a syringe as a handle, not as an aspirator.

Dr. Wentworth.—In answer to Dr. Caillé, who said that *turbid fluid* in meningitis was not a new discovery (or words to that effect), he misunderstood me. It was well-known that turbid fluid occurred in meningitis and was diagnostic, but my point was that the fluid was *never clear*, like the normal fluid, in meningitis, and that all the writers hitherto had stated that it often was so, especially in tubercular meningitis. This I denied and said it often appeared clear unless carefully examined, and that it always contained cells and fibrin.

I think Dr. Holt referred to the differential diagnosis of the various forms of meningitis and alluded to tubercle bacilli being found. I stated that the differential diagnosis could be made by cultures, by the different character of the cells in the exudation of tubercular meningitis and purulent meningitis, and by the inoculation of guinea-pigs with the fluid, the latter being a surer method of control in cases of tubercular meningitis, than by examining cover-glass preparations for tubercle bacilli. It was very important to have a sure method of control in order to learn whether tubercular meningitis was ever recovered from. So far as I know in the two or three cases of recovery which

have been reported, the diagnosis depended upon cover-glass preparations and there may have been a mistake in diagnosis.

With reference to albumin, I said there appeared to be a misinterpretation of the statement made in the German journals. They refer to the number of grammes of albumin in the litre and not to per cent. When they say "1.–2. pro mille," they mean 1.–2. grammes in 1000 c. c., which would be $\frac{1}{10}$–$\frac{1}{6}$ of one per cent. In the translations this has been referred to as 1.–2. per cent. In my quantitative tests for albumin I have found $\frac{1}{50}$–$\frac{1}{60}$ of one per cent. or less, in normal fluid and $\frac{1}{10}$–$\frac{1}{30}$ of one per cent. in the cases of meningitis thus far examined.

With reference to the danger of the operation, some one alluded to cases which had died after the operation and that it might be a dangerous procedure. I stated that the only fatal cases of which I was aware had occurred in cases of uræmia and cerebral tumor. In the former, one could hardly, with certainty, ascribe the death to the puncture. In cerebral tumor the operation often caused headache.

LOW TEMPERATURE PASTEURIZATION OF MILK AT ABOUT 68° C. (155° F.).

BY ROWLAND GODFREY FREEMAN, M.D.,

Pathologist to the Foundling Hospital; Pathologist to St. Mary's Free Hospital for Children, New York.

During the winter of 1891-92, stimulated by the thorough work of Bitter,[1] I made a study of the subject of pasteurization of milk. My conclusions at the time were[2] that pasteurization of milk at 75°C. (167°F.) was superior to other methods of sterilizing, and as there was no apparatus obtainable by which milk could be definitely pasteurized at that temperature, I set myself the task of devising one. The result exceeded my expectation. for I was able to contrive a simple apparatus which produced a sufficiently definite temperature without the aid of a thermometer. The use of a thermometer in sterilizing milk gives good results only when very carefully watched. It is moreover very difficult even when watching a thermometer to bring a fluid to any fixed temperature and hold it at that temperature for half an hour. The apparatus is based on the fact that if two fluids at different temperatures are placed in contact these two temperatures will be equalized. I found that by immersing in a definite amount of boiling water, the source of heat having been removed, a properly proportioned amount of cold milk introduced in bottles under such conditions that they will not break, I was able to raise the milk to about the desired temperature, i.e., 75°C. (167°F.). The amount of boiling water used in the apparatus was such that, in raising the temperature of the milk through about 60°C. (108°F.) the water itself lost an equivalent amount of heat so that when the milk reached the maximum temperature the water was of the same temperature.

It seemed to me wise at that time to arrange the apparatus for 75°C. because

a. The injurious chemical changes caused by heating milk are said to begin at 80°C.

b. Milk heated to 75°C. for 20 minutes is fairly sterile. Milk which before pasteurization contains a hundred thousand or more living germs in each cubic centimeter as a rule after pasteurization shows no growth on a Petri plate in three days.

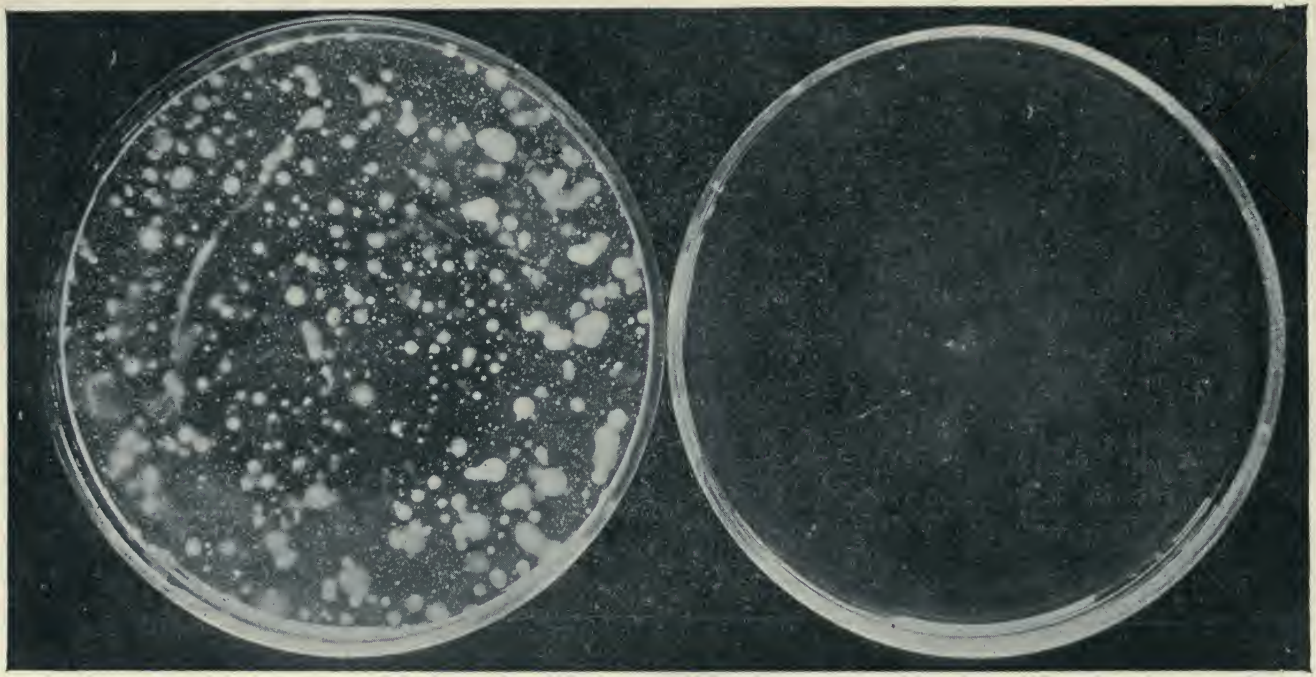
c. This temperature is sufficient to destroy the pathogenic germs which are most feared in milk including the bacillus tuberculosis, bacillus typhosis, and the bacillus diphtheriæ.

I felt convinced at that time that 75°C. was as high as milk should be sterilized and I was inclined to think that a lower temperature would be as safe were it not for the high temperature at which the thermal death point of the bacillus tuberculosis was at that time placed by those who had experimented to determine it. From one point of view there is decided advantage in pasteurizing milk below 70°C., for at about 70°C. the change in the taste of milk takes place, while milk heated to a lesser degree retains its original flavor unchanged. This change in the taste of milk caused by cooking, while it passes unnoticed by infants brought up on sterilized milk is often strongly objected to by older children and adults, so that in the practice of medicine we are frequently forced to a choice between raw milk or no milk at all.

If, then, milk can be satisfactorily pasteurized below 70°C. we have gained a distinct advantage in the taste of the milk and also avoid to a greater degree the chemical changes caused in the milk by heat.

Pasteurization at 68°C. as far as the ordinary air bacteria found in milk are concerned, gives almost as good result as pasteurization at 75°C. Usually a Petri plate of nutrient gelatine planted with pasteurized milk shows no growth for several days, while the same milk before pasteurization shows an extensive growth in 24 hours. The ordinary effect of the low temperature pasteurization is well shown by the accompanying photograph (Fig. 1). At the end of a week or ten days I find often a growth of only a single organism. This milk then is not absolutely sterile, but for that matter milk boiled at 100°C. (212°F.) a single time is not sterile.

The milk is, however, freed from almost all the living germs, and moreover those pathogenic bacteria which we know to be most likely to cause disease through their presence in milk are also destroyed. It will be seen that the only pathogenic bacterium in the accompanying table which is not destroyed by a short exposure to a temperature of 60°C. or less is the bacillus tuberculosis. There is evidence here that this micro-organism is destroyed by a temperature of less than 70°C. and indeed by a temperature of 60°C. of sufficient duration. These reports are the most recent on the thermal death point of the bacillus



PETRI PLATES ILLUSTRATING THE EFFICACY OF PASTEURIZATION AT 68° C. FOR THIRTY MINUTES IN DESTROYING THE BACTERIA OF MILK.

FIG. 1.—Photograph of a plate twenty-four hours after planting with 1 20 c. c. of raw milk. 7441 colonies are visible.

FIG. 2.—Photograph of a plate twenty-four hours after planting with 1-20 c. c. of the same milk after pasteurization for thirty minutes at 68° C. No colonies are visible.

tuberculosis that I have found. I will refer to these more particularly to show how thorough the work was which led to these conclusions.

TABLE OF THE THERMAL DEATH-POINT, IN A MOIST MEDIUM, OF CERTAIN PATHOGENIC BACTERIA.

SPECIES.	EXPOSURES.	OBSERVER.
Spirillum choleræ Asiaticæ.	60° C. for ten minutes.	Kitasato[3]
	59° C. for one minute.	Van Geuns[4]
	54° C. for five minutes.	"
	52° C. for four minutes.	Sternberg[5]
Streptococcus pyogenes.	52° C. for ten minutes.	Sternberg[6]
Bacillus typhosis.	60° C. for five minutes.	Buchner[7]
	60° C. for one minute.	Van Geuns[8]
	57° C. for five minutes.	Janowski[9]
	56° C. for ten minutes.	Sternberg[10]
	56° C. for five minutes.	Van Geuns[11]
Bacillus diphtheriæ.	58° C. for ten minutes.	Welch & Abbott[12]
Staphylococcus pyogenes aureus.	56° C.–58° C. for ten minutes.	Sternberg[13]
Bacillus coli communis.	60° C. for ten minutes.	Weisser[14]
Pneumococcus.	56° C.	Sternberg[15]
	60° C. for one minute.	Van Geuns[16]
Bacillus tuberculosis.	70° C. for one minute.	Grancher and Ledoux-Libard[17]
	70° C. for ten minutes.	Yersin[18]
	68° C.–68½° C. for twenty minutes.	Bitter[19]
	65° C. for fifteen minutes.	Forster[20]
	60° C. for twenty minutes.	Bonhoff[21]
	60° C. for fifteen minutes.	Schroeder[22]

In considering these data concerning the thermal death point of the bacillus tuberculosis it may be well first to point out certain reasons for variations in the results of different series of experiments.

a. Certain of the experiments were done with sputum, the thick masses of which are penetrated by heat more slowly than liquids.

b. Varying virulence in the bacteria used for inoculation.

c. In some, bovine tubercle bacilli were used while in others human.

d. The test of life of tubercle bacilli.

The bacillus tuberculosis is difficult to grow on culture media and its growth is, moreover, very slow. On this account in most of the experiments to determine the thermal death point of the organism, the test of life of the bacillus has been applied, not by planting in nutrient media, but by inoculation into susceptible

animals, preferably guinea-pigs. Such inoculations may be made either hypodermically, in the ear vein, or into the peritoneal cavity. At the time of the earlier experiments little was known of the lesions produced by dead tubercle bacilli. It has recently been shown by Prof. Prudden[23] that the injection of tubercle bacilli killed by repeated boiling and undoubtedly dead is followed by the production of lesions closely resembling some of the lesions caused by the introduction of living tubercle bacilli. After such injection an examination of the organs showed circumscribed collections of epithelioid cells and giant cells. In this tissue tubercle bacilli were found. These lesions were apparently due merely to the mechanical irritation of the dead tubercle bacilli and there was no indication that any general progressive disease was set up. This work is of great importance in connection with the determination of the thermal death point of the bacillus tuberculosis for in every such experiment either living or dead tubercle bacilli are injected, so that any observer not familiar with the action of dead tubercle bacilli might pronounce an animal tuberculous that was suffering only from the mechanical irritation of dead tubercle bacilli and as a result would set the thermal death point of the tubercle bacillus higher than it should be.

Yersin,[24] working in the Pasteur Institute in 1888, in a series of experiments with an old culture of tubercle bacilli found that he could get a growth after an exposure of ten minutes to a temperature of 60°C. but that after an exposure of the same duration to 70°C. no growth was obtained. These experiments were repeated a number of times with the same result.

Bitter,[25] of the Hygienic Institute of Breslau, in an article published in 1890, states that 68°-68.5°C. for 20 minutes will destroy the bacillus tuberculosis. These results were obtained by inoculation experiments with guinea-pigs.

Prof. Forster,[26] of the Hygienic Institute at Amsterdam, published in October, 1892, the result of a long series of experiments to determine the thermal death point of the bacillus tuberculosis. He used inoculation experiments in guinea-pigs with various materials including a milky substance squeezed from tuberculous tissue of the udder, also tuberculous tissue from the pleura and sputum containing the tubercle bacillus. He thus made use of both the human and bovine tubercle bacillus. He

found that they were killed by 55°C. for 6 hours, by 60°C. for one hour, but were not killed by 60°C. for 45 minutes.

This article was followed two months later by one by Dr. Bonhoff,[27] an assistant of the Hygienic Institute at Berlin, giving the results of experiments extending over the past six months. He used a culture of the bacillus tuberculosis and injected guinea-pigs. He says "from these results I conclude that about a temperature of 60°C. for 20 minutes is ample to kill tubercle bacilli in pure culture, or at least to render them harmless to the animal organism." This publication of Dr. Bonhoff places the thermal death point lower than any previous observation.

A year later Prof. Forster[28] published the result of another series of experiments in his laboratory, done by C. de Man,[29] to determine more accurately the time necessary to destroy the organism at different temperatures. The data of this second paper correspond fairly with that of the first, but are fuller. He finds that the following exposures will kill the bacillus tuberculosis:

55° C. for four hours.
60° C. for one hour.
65° C. for fifteen minutes.
70° C. for ten minutes.
80° C. for five minutes.
90° C. for two minutes.
95° C. for one minute.

Schroeder's[22] figure is of itself not of much value since it was the result of only one experiment, but taken with the other evidence presented is confirmatory. Schroeder undertook to ascertain by the inoculation of guinea-pigs the extent of the occurrence of the bacillus tuberculosis in the milk supply of Washington. He proposed to subject the milk before injection to a temperature of 60° C. for fifteen minutes to destroy other bacteria which might interfere with his results, supposing that such temperature was not sufficient to kill the bacillus tuberculosis. To make sure that such exposure would not injure the bacillus tuberculosis, he planted milk from a culture of the bacillus tuberculosis, and then used a temperature of 60° C. for fifteen minutes. The animal injected with the milk was killed on the fifty-eighth day and showed no evidence of tuberculosis, although two other animals subjected to the same treatment without the heating died on the twenty-fifth and twenty-eighth days.

The evidence of these observers seems to me sufficient for a conclusion that a temperature of 65° C. for fifteen minutes is sufficient to kill tubercle bacilli. We have, so far as I know, no equally recent work the results of which are opposed to this conclusion. Having been thus persuaded that the only obstacle to the pasteurization of milk below 70° C. has been removed, I have modified the pasteurizer in order to fit it for the desired temperature. In so doing I have been able to do away with a source of error in the old apparatus, and so far as I know in all sterilizers, that is, a marked difference of temperature between the milk in the bottom and that in the top of the bottle. By elevating the receptacle so that only the lower portion is in contact with the hot water this equalization of temperature through-

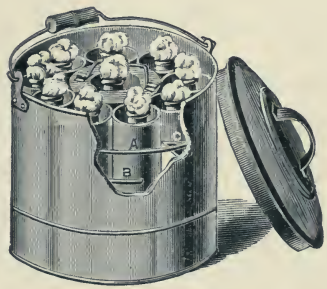


FIG. 3.—Showing the apparatus arranged for heating the milk before the pail is covered.

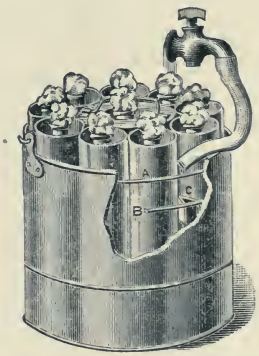


FIG 4.—Showing the apparatus arranged for cooling the milk.

out the bottle is obtained. This apparatus is so arranged that a temperature of more than 65° C. and less than 70° C. may be maintained for half an hour.

If the milk introduced has a temperature of 10° C. (50° F.) the resultant temperature will be about 68° C. (155° F.); while if the milk has a temperature of 20° C. (68° F.), the resultant temperature will be about 69½° C. (157° F.). Milk from a good refrigerator has usually a temperature of about 15° C. (59° F.).

The apparatus (Fig. 3) consists of two parts, a pail for the water and a receptacle for the bottles of milk. The pail is a simple pail with a cover; there is a groove extending around the pail to indicate the level to which it is to be filled with water, and supports inside for the receptacle for the bottles of milk to

rest on. The receptacle for the bottles of milk consists of a series of hollow zinc cylinders fastened together; this fits into the pail so that the lower inch of the cylinders is immersed in the water. This receptacle has two sets of horizontal supports, the upper set continuous around the receptacle for use while the milk is being heated; the lower interrupted set is used for raising the receptacle during cooling. Such receptacles are made for ten 6-oz. bottles, seven 8-oz. bottles, three 1-pint and one ½-pint bottles and two quart bottles. There is also a large apparatus for the use of hospitals or public institutions which has a receptacle for forty-three 6-oz. or 8-oz. bottles.

The apparatus is used in the following way: The pail is filled to the level of the groove with water, covered and put on the stove, the receptacle for the bottles being left out. The bottles of milk are then filled, stoppered with cotton and dropped into their places in the cylinders. Sufficient water is poured into each cylinder to surround the body of the bottle. As soon as the water in the pail boils thoroughly it is taken from the stove and set on a mat or table or other non-conductor in a place where there is not a draft of wind blowing on it. The lid of the pail is removed and the receptacle for the bottles of milk is put in the pail so that the receptacle rests on the upper continuous supports. The lid is then rapidly put on the pail and the pail is thus allowed to stand for three-quarters of an hour. During the first fifteen minutes the temperature of the milk rises—as may be seen by the accompanying chart—to about its maximum or above 65° C., the point desired for pasteurizing, and remains there the remaining thirty minutes. During the last fifteen minutes it falls about one degree; at the end of forty-five minutes the cover of the pail is removed, the receptacle is lifted and given a turn so as to rest on the upper supports (Fig. 4), thus bringing the top of the cylinders containing the bottles above the level of the pail. The pail is then put under a cold water faucet and the water is allowed to run into the pail and overflow, but it should not run into the cylinders. Thus the hot water is replaced by cold water, and in fifteen minutes the milk in the bottles is of about the temperature of the cold water used. The bottles may then be put into a refrigerator until required for feeding. This rapid cooling is a most important part of a low temperature sterilization, the importance of which is apt to be overlooked.

Certain points in this chart to which I wish to call special attention are:

1. The apparent lack of precision in the action of the apparatus, due to the unknown temperature of the milk introduced, is to a considerable extent corrected. The amount and tempera-

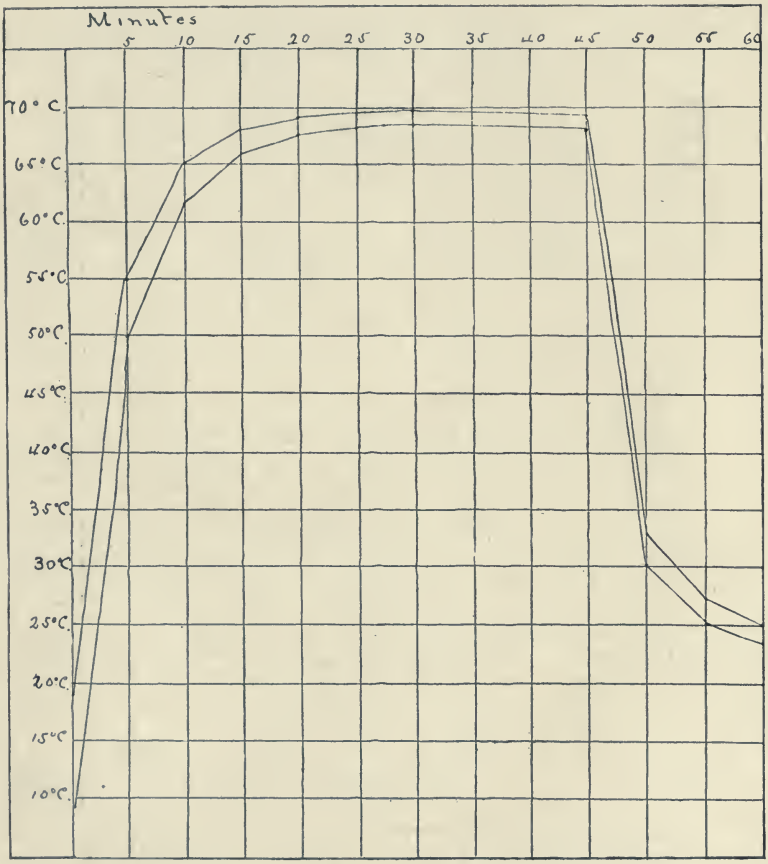


CHART SHOWING TWO OBSERVATIONS ON THE TEMPERATURE OF MILK DURING PASTEURIZATION IN THE APPARATUS.

ture of the boiling water used for heating is definite; the amount of cold milk to be heated is definite, but the temperature of the milk is such as may be covered by the word cold or by refrigerator temperature. The chart shows that the apparatus will correct a considerable variation of the temperature of the milk

used by the ability of the boiling water to carry cold milk through a greater number of degrees of temperature than warmer milk; on this account whether the milk is introduced at a temperature of 10° C. (50° F.) or 20° C. (68° F.), the resultant temperature varies only 2° C.

2. The very rapid rise in the temperature of the milk introduced. It rises about thirty-five or forty degrees in the first five minutes, about ten degrees in the second five minutes and about five degrees in the third.

3. The even temperature preserved after the rise; a variation of not more than a degree during the last twenty-five minutes.

4. The rapid fall of the temperature in a cold water bath; a fall of about thirty-five degrees in the first five minutes. This cooling in a cold water bath takes place eight times as fast as in a refrigerator.

SUMMARY.

Pasteurization at between 65° C. (149° F.) and 70° C. (167° F.) is recommended for the following reasons:

1. It destroys almost all the ordinary air bacteria which occur commonly in milk.

2. It destroys the bacillus tuberculosis, the bacillus typhosis, the bacillus diphtheria and many other pathogenic bacteria.

3. It causes no change in the taste of the milk and avoids those chemical changes in milk which are produced by higher temperatures.

4. It is possible to pasteurize accurately at this temperature without the use of a thermometer.

REFERENCES.

1. Bitter. Zeitschrift fur Hygiene, 1890. Bd. VIII. S. 240.
2. Freeman. Medical Record, 1892, July 2d.
3. Kitasato. Zeitschrift fur Hygiene, 1888. Bd. V. S. 134.
4. Van Geuns. Archiv. fur Hygiene, 1889. Bd. IX. S. 369.
5. Sternberg. Manual of Bacteriology. P. 504.
6. Sternberg. Ibid. P. 276.
7. Buchner. Centralblatt fur Bacteriologie, etc., 1888. Bd. IV. P. 386.
8. Van Geuns. Ibid.

9. Janowski. Centralblatt fur Bacteriologie, etc., 1890. Bd. VIII. S. 417.
10. Sternberg. Ibid. P. 351.
11. Van Geuns. Ibid.
12. Welsh and Abbott. Bul. of Johns Hopkins Hospital, 1891. Vol. II. P. 25.
13. Sternberg. Ibid. P. 267.
14. Weisser quoted by Sternberg. Ibid. P. 442.
15. Sternberg. Ibid. P. 297.
16. Van Geuns. Ibid.
17. Grancher and Lidoux-Libard. Archiv. de Med. Expr., etc., 1892. IV. 1.
18. Yersin. Annales de Inst. Pasteur. I. 2. P. 64.
19. Bitter. Ibid.
20. Forster. Hygienische Rundschau, 1893. No. 15.
21. Bonhoff. Hygienische Rundschau, 1892. No. 23.
22. Schroeder. Bul. of U. S. Bureau of Animal Industry. No. 7. P. 75.
23. Prudden and Hodenpyl. N. Y. Medical Journal, 1891, June 6 and 20.
24. Yersin. Ibid.
25. Bitter. Ibid.
26. Forster. Hygienische Rundschau, 1892, Oct. 15. S. 869.
27. Bonhoff. Ibid.
28. Forster. Hygienische Rundschau, 1893. No. 15.
29. C. de Man. Archiv. fur Hygiene. Bd. XVIII. 2 Heft.

203 WEST FIFTY-SEVENTH STREET.

DISCUSSION.

DR. T. M. ROTCH.—These experiments of Dr. Freeman's are very important and practical. I hope that he will also show us how to heat a single bottle when it is to be heated to 99° or 100° F. I suppose it could be done in the same way. As to the experiments which Dr. Freeman reports, I have had something of the same kind done in Boston with very similar results.

DR. L. EMMETT HOLT.—I have used Dr. Freeman's apparatus for years with the higher temperature (167° F.), and have found it most satisfactory. It is so simple that any mother or nurse can use it. The chances of error are very small if the directions

are followed. The importance of rapid cooling is often forgotten. Although nurses are told, they will not do it unless the point is emphasized.

DR. J. P. CROZER GRIFFITH.—I believe, even without personal experience with it, that Dr. Freeman's pasteurizer is one of the most useful appliances we can have in the preparation of food for infants. I have seen it before but have never used it, although I shall certainly do so in future. It is not as widely known in Philadelphia as it deserves to be—in fact, is very little known there.

I have experimented with pasteurizing with an ordinary sterilizer, leaving the cover off to a greater or less extent, and using a thermometer in the milk to determine how accurate the results were. In this way a fair result can be obtained if the process is carefully watched. The trouble is, however, that the heat of the stove, and the consequent temperature of the milk, is sure to vary at different times, and that accuracy is impossible even with the strictest attention.

Dr. Freeman's pasteurizer obviates all those difficulties, and is besides so simple in its application that I have only words of praise for it.

DR. FREEMAN.—Dr. Rotch's suggestion to apply the same principle used in the pasteurizer to the heating of a single bottle to the proper temperature for feeding, I will try to carry out. It would seem an easy matter to adjust such a bottle-warmer so that it would produce an accurate temperature.

A question has been raised as to the advisability of the term pasteurize. It seems to me that this name is necessary, as no other word indicates the same thing—that is a low temperature sterilization followed by rapid cooling. The rapid cooling is a most important part of the process. If we use the expression low temperature sterilization, the rapid cooling is apt to be overlooked.

Milk, should be used only during the twenty-four hours following pasteurization. Although the pasteurized milk will not sour in several days if kept cold, it should be used only during the interval I have indicated. Bottles of milk pasteurized at about 68° C. and left standing on my laboratory table during the spring usually showed no separation of casein in less than three days. Milk pasteurized at 75° C. I have found to keep for a week or ten days in a refrigerator. A very good demonstration of the keeping qualities of pasteurized milk has been afforded by the Nathan Straus Milk Depots of New York. The milk sold at these depots is pasteurized at about 75° C. in large apparatuses constructed on the same principle as the one I have just shown. After cooling it is stored in iced water until dispensed. As many as seven thousand bottles are distributed by these depots during

some days in summer. This milk supplies the very poor of New York, and goes into many homes that are not supplied with ice. Two years ago, while preparing a paper I inquired of the superintendent whether they were at all troubled by any of the milk souring in the tenement houses. He replied that they had had one complaint, which he had investigated, and had found that the milk had been kept under the kitchen stove. When this charity was started in 1893 I advised pasteurization at 75° C., and it was undertaken, although the gentleman having charge of it was assured by others that milk pasteurized at this temperature would not keep under the conditions existing in tenement houses, and that a temperature of at least 80° C. or 90° C. should be used. They have seen no necessity for using a higher temperature after three years' experience.

TUBERCULAR ABSCESS OF THE BRAIN.

BY SAMUEL S. ADAMS, M.D.,

Professor of Pediatrics, University of Georgetown; Physician to the Children's Hospital, Washington, D.C.

H. C., colored, male, aged ten years, was admitted to the Children's Hospital, March 22, 1895. His father had died of phthisis, but his mother was in good health. He had had malarial and typhoid fevers. Since the attack of typhoid fever, about a year ago, he has been very much debilitated, and has frequently complained of severe pains in his eyes, usually at night. He is poorly nourished and anæmic. There is much weakness of lower extremities and slight muscular atrophy.

The head is greatly elongated and is prominent at the vertex. There is hebetude and mental deficiency. His movements are slow, but reflex action is not impaired. Says he has pains in his head and eyes all the time. Eyes exophthalmic, and he says he can distinguish objects, which is very doubtful from his answers to questions. There is choroiditis and choked disc. Paralysis of the oculo-motor. Speech is slow and distinct, his responses being accurate but slow.

March 26. He is dull and quiet most of the time. At times he keeps up a rotary motion of the head. Profuse salivation. Had a convulsion to-day, characterized by tonic spasm on the right side and clonic on the left; consciousness was only slightly impaired. This condition lasted five minutes and was followed by sleep lasting an hour.

April 12. Had a convulsion this morning which began in the fingers of the left hand, gradually involving the entire limb, and extending to the lower extremity. Right side slightly affected. No change in mental state. Expectorates profusely. Convulsions invariably clonic.

May 30. Convulsions irregular in frequency, but have not changed in character.

His condition gradually grew worse until September 26, when left hemiplegia was noted. Cutaneous hyperæsthesia marked. Screams with starting pains, particularly severe at night. Head seems to be enlarging in antero-posterior diameter and eyeballs more prominent.

December 23. Patient weaker and sleeps most of the time. If placed in a position he maintains it until some one moves him. Usually lies curled up. He sees devils, spirits, and angels. Shrieks at the top of his voice; is very irritable and cries at the slightest touch. He has a fluent vocabulary of profanity which is easily extracted. The Resident reported that his mind was almost gone; that his profanity annoyed the nurses; and that he was very filthy about his person. Thinking it might be well to reprimand him in order to test his mental capacity, I told him that it was wrong to swear at the nurses who were so kind to him. "Dr. C. says you swear at your friend, Miss D." He answered emphatically but slowly: "Dr. C. is a d——d liar"—an evidence that the mental faculty was not entirely useless.

January 17, 1896. He has gradually grown worse and about January 1st tubercular softening of the lung was diagnosticated. All the symptoms of pulmonary tuberculosis developed and he died to-day.

Necropsy by Dr. D. S. Lamb.—Right half of brain showing rather firm nodular masses, varying in size, in the cerebral hemisphere, surrounded by much softening. In the recent state the condition was as follows: adhesions of membrane to skull over superior frontal lobe: yellowish flocculent space involving upper part of operculum and upper frontal gyrus, altogether 2.5 by 2 by 1.5 inches, and which on section showed yellowish turbid liquid within ragged cheesy walls: a fine whitish mottling over supramarginal and posterior part of upper temporal gyri, which on section showed firm cheesy masses and yellowish watery liquid. Almost all the hemisphere, especially its anterior part, was quite œdematous. The broken down substance involved the lateral ventricle: fornix quite soft. The left hemisphere was somewhat congested: lateral ventricle filled with bloody serum.

Hypertrophy and Atrophy of Skull.—Two portions of skull including skull-cap and bones of upper part of face. Sagittal suture 5.25 inches long, coössified except in its anterior inch; part of left coronal suture coössified; atrophic openings in right coronal suture; 13 similar openings in lower part of right parietal bone; 3 small similar openings along line of sagittal suture; one similar opening near right frontal eminence; parietal bones above parietal eminence irregularly thickened; orbital plates heaped up far above level of cribriform plate and show similar atrophic

openings; lesser wings of sphenoid not united to frontal; olivary processes not well marked; atrophic openings in great wings; right antrum of Highmore less excavated than left; Wormian bones in each spheno-malar suture; and nasal bones one inch long in median line.

Heart.—Quite a large amount of straw-colored serum found in the pericardium, otherwise normal.

Lungs.—Many miliary tubercles seen and felt in lower lobe of left; a good deal of blood and some serum, with small tubercles throughout—apparently an acute condition—in upper. The entire wall of the right side shows adhesions of pleura both old and recent; the upper surface of the diaphragm on right is covered with miliary tubercles. Tubercles were found in the mediastinal space. Bronchial glands enlarged and on section show tubercles and caseous masses. Adhesions between lobes of right. Yellowish cheesy masses throughout and one small cavity—chronic—in lower right lobe. The upper shows same condition, but is more grayish and somewhat pneumonic.

Liver.—Fatty and has appearance of nutmeg.

Spleen.—Large and full of cheesy tubercles. Splenic glands along pancreas enlarged and cheesy.

Kidneys.—Both enlarged and congested.

Intestines.—Ulcers all through the lower part of the ileum near the ileo-cæcal valve. Vessels are injected and small tubercles are seen and felt in the last foot of ileum. One adhesion of bowel just under an ulcer near cæcum. Mesenteric glands enlarged and caseous.

Dr. Lamb writes: "The tumors of brain in the boy with general tuberculosis show only a few giant cells, the most of the mass being without any recognizable structure. Drs. Reed and Gray agree with my original opinion that the masses are probably of tubercular origin."

COMMENTS.

From the history and the condition of the boy on admission I was satisfied that there was a tumor in the brain, but an opinion as to its nature was never formed. He was at first placed upon specific treatment but he was never benefited by it. Attempts at localization proved futile.

1 DUPONT CIRCLE.

MALIGNANT ENDOCARDITIS.

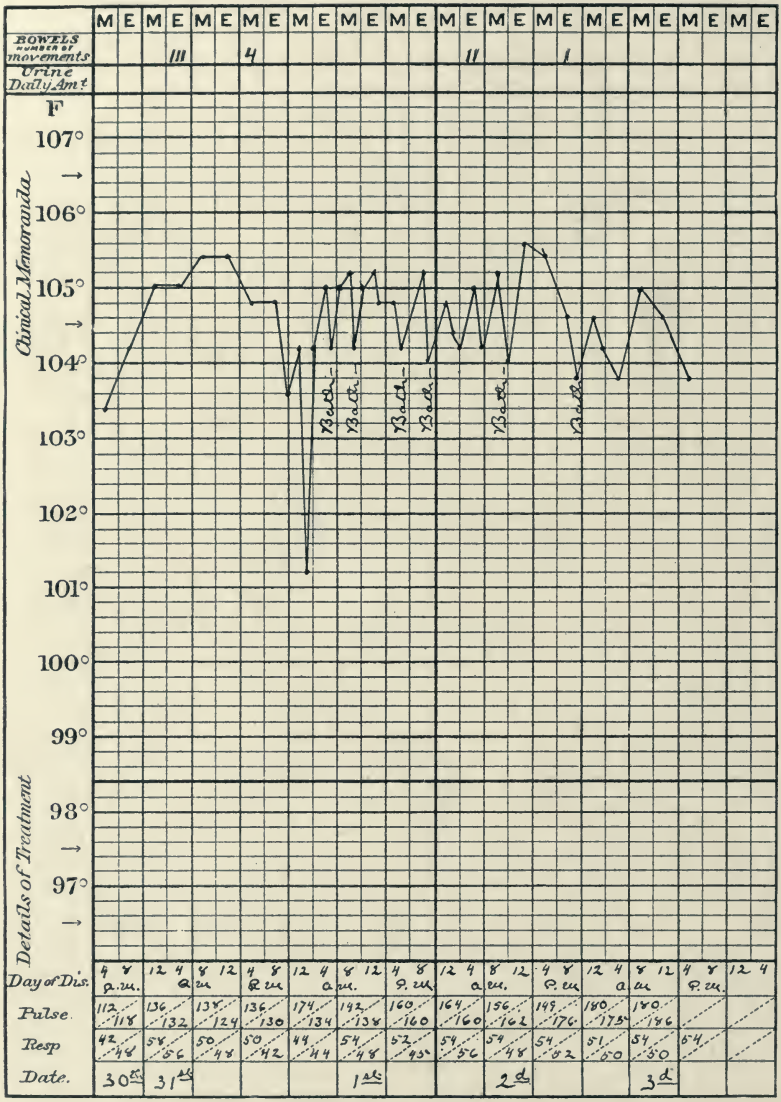
BY J. HENRY FRUITNIGHT, A.M., M.D.

Fellow of New York Academy of Medicine of American Academy of Medicine; Visiting Physician to St. John's Guild Free Hospital for Children, New York, etc., etc.

Introductory to the main portion of this paper I shall detail the clinical history and *post mortem* findings of a case of this disease which recently came under my care.

On January 30th of this year, Rosina K., aged eleven years, was brought to my office by her father, with an indefinite history of illness of a week's duration. She had a temperature of 105° and presented the general appearance of a patient ill with typhoid fever. On the same day I had her admitted to my service at the St. John's Guild Free Hospital for Children, in New York City. Here a more careful examination of the patient was made. It was learned that she had had measles and pertussis, and that two years previously she had had acute articular rheumatism, both ankle joints being especially painful and swollen. The father also had been attended by me, some years ago, for two very severe attacks of acute articular rheumatism. The girl had sickened a week previous to her admission to the hospital with vomiting, headache and a high fever. Since then she has become drowsy and continues to complain chiefly of headache. She has anorexia, and is greatly prostrated. She is without pain. Her tongue is fissured and covered with sordes. The bowels are constipated. No rose-colored spots can be found. The abdomen is tympanitic and tender. The spleen is enlarged and tender. The lungs are normal. The physical examination of heart gave negative results, and no murmur was detected. Her temperature on admission was 103½°, pulse 112, and respirations 42 per minute. The case was supposed to be one of typhoid fever.

On January 31st, the Brand bath was ordered, and to be repeated every four hours if the temperature indicated its employment. The bath produces but a slight fall in the temperature, and three-quarters of an hour later the fever has risen to the same high degree registered before the administration of the bath. The patient is now delirious. She lies with her eyes half-closed and moans continuously. On February 2d, a large bulla filled with a hemorrhagic fluid appeared on the first joint of one of the



TEMPERATURE RANGE.

In Case of Malignant Endocarditis.

great toes. An ecchymosis has also appeared on the right elbow. The heart sounds are feeble and continuous. On February 2d the pulse has run up to 176 beats per minute and is very feeble, and in the night the pulsations reached 180 per minute. The patient is comatose. A number of petechiæ have appeared on the body and extremities. A few sub-conjunctival hemorrhages have also been observed. The pulse continued to grow weaker and the dyspnœa increased proportionately, the nostrils being drawn in at every inspiration. At half past five in the afternoon the patient died in a state of coma.

The treatment had been in the main a stimulating one, embracing the frequent and liberal administration of whiskey, reinforced by digitalis and hypodermatic injection of strychnine and supplemented with a generous diet of milk and beef juice. As already stated, the fever was combated with the Brand system of baths and ice bags to the head for the relief of the cephalalgia. The chart exhibiting the temperature range, with the number of respirations and pulse beats, will be found elsewhere.

Seventeen and a half hours after death an autopsy was made upon the body by the pathologist to the hospital, Dr. Frank Grauer, with the following results: Rigor mortis was present. There was a slight abrasion over the instep of the left foot which had suppurated and was partially cicatrized. Petechiæ were present over abdomen, about elbow and on lower extremities, also in the conjunctivæ. The lungs were normal. The heart exhibited compensatory hypertrophy, showed small subserous hemorrhages and was found to have stopped in diastole. The right ventricle was dilated and contained fluid blood. Small vegetations and ulcerations were present on the tricuspid and mitral valves. The heart weighed six ounces.

The kidneys showed thrombotic infarctions and each one weighed three ounces. The suprarenal capsules were slightly adherent. The spleen weighed eight ounces. The liver showed slight parenchymatous hapatitis and weighed two pounds and three ounces. The mesenteric glands were somewhat enlarged.

The intestines exhibited externally hemorrhagic spots and, on the interior, ulcerations, but not those of typhoid fever.

In the left lumbar region there was circumscribed peritonitis. In the stomach submucous hemorrhages were present and some ulcerations were found.

The bladder was very much thickened, and revealed submucous hemorrhages. The uterus and ovaries also showed small hemorrhages.

The brain was highly congested. Bloody fluid was found in the ventricles. A hemorrhage into the arachnoid covered the first frontal and orbital convolutions on the right side.

The microscopical examination, also made by Dr. Frank Grauer, the pathologist to the hospital, shows that the vegetations consist mainly of granular fibrin with nuclei of round and

spindle-shaped cells scattered about in the lower segment. The subjacent endocardial tissue is infiltrated with a large number of round cells which extend down to the muscular fibres revealed in the specimen. The vegetations contain a large number of micrococci which are arranged in groups and which extend through the fibrous tissue to the heart wall. A plate culture made from the vegetations shows almost pure colonies of the staphylococcus pyogenes aureus.

The pathological anatomical diagnosis of the case is therefore malignant endocarditis, due to streptococcus infection. A few remarks upon the medium and source of infection are *apropos.* Though no cardiac murmur or lesion could be detected by physical examination, the presumption is nevertheless tenable that, inasmuch as the patient had passed through an attack of rheumatism, some change, possibly slight, must have taken place in the endocardium. This changed condition was then the predisposing cause, and very possibly the small abrasion on the foot of the patient was the source whence the streptococci secured entrance into the circulation, and in consequence exerted their baneful influence upon the already affected endocardial membrane; the micro-organism thus acting as the exciting cause of the disease in this case.

For most of our knowledge concerning malignant endocarditis we are indebted to Weichselbaum, Wissokowitsch, Grancher, Babes, Netter, and Osler. Weichselbaum has demonstrated that this disease always results from an infectious process which has followed an assault upon the endocardium by some variety of microbe.

Although so large a number of micro-organisms are known to exist, and notwithstanding the many conditions of the blood which may engender these various microbes, malignant endocarditis is not so prevalent as might be anticipated.

For the development of a malignant endocarditis the micro-organism must first gain access to the blood, which is not always usual or easy, and besides a susceptibility to lesion on the part of the endocardium must also pre-exist. In all likelihood the disease is brought directly by the blood to the valves and not through the capillary channels. Sometimes the point of entrance of the microbes cannot be determined in such cases; very likely they have gained an entry through the respiratory tract.

The injection of a pure culture of the *staphylococcus pyogenes aureus* directly into the circulation of an animal whose cardiac

valves have been irritated with a probe, has produced the signs of a malignant endocarditis.

The right heart is very infrequently affected in comparison with the left side of the organ, notwithstanding the fact that the infected matter usually first passes through the cavities of the right side.

It has been proven that micro-organisms have a predilection for blood rich in oxygen, and hence more favorable conditions exist in the left side of the heart for the activities of the microbes.

Microbes are met with in every case of endocarditis. They are not always, nor are they all, of the same variety. Frequently several kinds of bacteria are associated in the same case of endocarditis. Nor is endocarditis produced by a specific microbe, as are tuberculosis, tetanus, diphtheria and the like. The pyogenic varieties of micro-organism seem to prefer the mitral valve for their point of attack, whilst the pneumococcus selects the aortic for its assault.

The disease is much more virulent and death follows very rapidly when the infection is due to the streptococcus. When due to the staphylocccus the course of the disease is less rapid. As the disease is, in the majority of cases, dependent upon a mixed infection caused by the combination of different microbes, the symptoms will be mixed and not at all clearly defined, rendering the diagnosis difficult and well-nigh impossible. A commingling of the several kinds of bacteria renders their action more violent than when a single variety is operative, and likewise when the vitality of their host is depreciated the pathogenic activity of the microbes is augmented. Groups of micrococci have been demonstrated in the skin lesions of malignant endocarditis by Bramwell.* Malignant endocarditis may be primary, affecting the lining membrane of the heart or of its valves from the outset of the disease and may be restricted to these parts. Again, it may be secondary to an acute rheumatism, or pneumonia, or to various specific fevers; or, finally, it may be allied to associated septic processes.

As already intimated the disease is essentially of a septic character. The local and constitutional symptoms and effects are evolved through the transportation in the circulation of morbid products to different parts of the body.

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* International Journal of Medical Science, July, 1886, page 28.

The disease is a rare one, though many cases which may have been classified as typhoid or typhus fever, in which the symptoms may not have been unequivocal, might possibly have been cases of malignant endocarditis. When the disease is primary no complicating disease or lesion can be discovered. This form is very rare indeed. The secondary malignant variety is not so very frequent in the course of a rheumatic endocarditis and it is extremely rare in chorea. This form is of most frequent occurrence during the course of a pneumonia, and is under these circumstances of a most malignant type.

It has complicated a small proportion of cases of cerebro-spinal meningitis. It accompanies tuberculosis very rarely. It has been found in cases of diphtheria, small-pox and scarlet fever. It has been claimed to occur in the course of an intermittent fever, but this is undoubtedly an error, for the alternating pyrexia and apyrexia do not depend upon the specific plasmodium, but upon the malignant endocarditis. The disease has also been met with in cases of epidemic dysentery.

The scantiness of material for observation makes it very difficult to determine the duration of an attack of the disease, and besides, the beginning of the attack is not always exactly defined.

Many cases have been reported which confirm the possibility of infection through slight traumatisms, as scratch of pin, pulling off a hangnail, and so forth. The case reported at the beginning of this paper appears to have been of this description.

The disease occurs rarely in children, probably because it is so specially connected with degenerating influences, such as alcoholic excesses, want, exposure, etc. The first case occurring in a child was reported by Dr. Kirkes about forty years ago. The patient was a boy fourteen years of age. Prof. Osler, as reported in his Gulstonian lectures on the subject, delivered in 1885, and published in the London *Lancet,* pp. 415, 459, 504 of that year, had up to that time seen but three cases in children under ten years old. Dr. H. B. Donkin, in his book on "Diseases of Children" (p. 413, Ed. 1893), states that "he has seen but three probable cases of the disease, of which only one was verified by autopsy." Dr. Donkin adds that an autopsy is the only proof of the diagnosis of this disease. Cheadle says that in the records of the Hospital for Sick Children, London, England, to which only children under twelve years of age are admitted, for a period of twenty years but one case is to be found. Though I

have met with three adult cases in my own practice, the case herewith reported is the first I have encountered in children.

The lesion of malignant endocarditis may be vegetative, ulcerative, or suppurative, or it may be a combination of all these processes. Destruction of endocardial tissue and even necrosis may accompany the vegetations. The ulcerations may be superficial or deep even to the extent of perforation. The vegetations are made up of granular and striated fibrin harboring colonies of micro-organisms.

The endocardial membrane, situated at the base of these vegetations, shows infiltration and proliferation. The destruction of tissue follows the gradual extension of the necrobiotic process present in the endocardium.

The ulcerative inflammatory processes present in the endocardium may give rise to minute emboli, or these may even result from the intensity of the inflammation and exudations. In the course of necrosis portions of dead tissue may become loosened to be swept away in the blood current. Thus originate the embolic infarcts found in the spleen, brain, kidneys, and other organs. Sometimes minute abscesses will be found on the curtains of the valves. Malignant endocarditis has been observed in conjunction with osteo-myelitis. Osler has shown that in septic endocarditis the mitral valve is involved in the ratio of sixty-seven cases to fifty-three cases of aortic involvement, and that in three-fourths of the cases of malignant endocarditis, a valvular lesion had pre-existed. Osler also states that the typhoid type of the disease is the most common, yet in many of these cases no source of infection can be traced.

The malignancy of the disease is due to and depends upon the peculiar underlying pathological state which has superinduced the malignant endocarditis.

The symptoms of malignant endocarditis are apt to be very obscure. They may simulate those of very different pathological conditions, as well as vary in different individuals.

The symptoms may be divided into two predominating types: first, the pyæmic or septic, and secondly, the typhoid. A lesser number of cases resemble in their semeilogy an intermittent fever. In some cases the symptoms point to so severe an involvement of the cerebro-spinal system and are so characteristic, that the diagnosis of acute cerebro-spinal meningitis may be made. The symptoms are partly constitutional, caused

by a mingling with the blood of detritus derived from the disintegrating valve, and partly local, produced by emboli which hinder the functions of the particular organ which they may have invaded, as shown by the cutaneous petechiæ and the infarctions met with in the various organs.

The typhoid type of the affection is undoubtedly the most common. The curving exacerbations may simulate those of typhoid fever. The temperature curve is prone to be irregular, however, and not typical. It will show remissions, but these are not rhythmical or regular.

In those cases in which the symptoms assume the septic or pyæmic type there pre-exists some external wound or point of suppuration, or area of necrobiosis whence the disease takes its origin. These local lesions may be very insignificant, as has been said on a previous page. Such cases have sometimes been designated infectious endocarditis.

The disease may be very rapid in its irruption and course, with intense fever maintained to its termination. Again, it may be slow in its progress, with intermissions of fever, even having a period of apyrexia, only to again suddenly rise, the disease advancing by leaps and bounds. These exacerbations of the disease are very likely owing to a general infection of the blood caused by the periodical loosening of infectious particles from the endocardium in the course of the ulcerative process present in that membrane. In the pyæmic type the fever partakes of the hectic character, accompanied by rigors and profuse perspiration. There is profound prostration, with a rapid, feeble. incompressible pulse. More or less diarrhœa prevails. The spleen enlarges. Sometimes jaundice supervenes. The abdomen is distended. The urine is dark, containing albumin and sometimes blood. The passage of urine and faeces become involuntary. In the stools blood is frequently present. Petechiæ soon appear. These are found chiefly on the abdomen, less often on the face and the extremities, though they may be present over the whole surface of the body. A rapidly developing pustular exanthem has also been observed. An erythematous and a papular eruption have also been encountered. The joints are usually painful. Patients complain of giddiness and headache. In most cases delirium and coma, indicative of meningeal or cerebral complication, close the scene.

When a patient is free from cardiac disease, malignant endo-

carditis may be very insidious in its coming, and may thus remain undetected.

The disturbances of respiration which are met with in many cases are very likely produced by obstructions in the pulmonary circulation. Very infrequently these obstructions may be so serious as to induce hæmoptysis in consequence of the profound stasis caused by this interference with the pulmonary circulation.

In many cases during the entire course of the disease, the physical examination of the heart may be negative in its results, yet the autopsies held in such cases reveal the presence of extensive lesions about the valvular openings. Such has been known to be the fact when the lesion at the mitral orifice was very grave and must have given rise to considerable regurgitation. The disease therefore can be present, unaccompanied by any adventitious sounds or murmurs. Many such cases are recorded in the literature of the subject. The case reported in this paper was of this description. In a few cases the cardiac symptoms are most prominent, for occasionally a patient may, early in the disease, complain of oppression over the epigastrium and of dyspnœa accompanied by a feeling of dread and anxiety and palpitation of the heart.

Complaints of definite local discomfort calling attention to the heart are seldom made, however, even though a cardiac lesion may co-exist. It may be laid down as a general rule that we never find an enlarged area of cardiac dullness unless chronic valvular disease be present or a fresh pericardial exudation has taken place. In some cases where the valves are the seat of trouble, no murmur will be audible during the *entire* progress of the case.

In most cases, however, a systolic blowing sound is heard early in the course of the disease. If an aortic bruit be heard it may be assumed to favor the diagnosis of a malignant endocarditis.

To make the diagnosis of malignant endocarditis is in most cases very difficult. It will sometimes be impossible to differentiate it from other diseases. In fact it has been very seldom diagnosticated with certainty in life. It has generally either been overlooked or supposed to be present. This is not at all surprising when we consider how little the patient may complain locally, and how ambiguous in their significance both the local

and constitutional symptoms may be. There seems to be no fixed relationship between the clinical features of the disease and the pathogenic agencies producing it and its lesions. As has been remarked, the local cardiac symptoms are so infrequently present that the diagnosis cannot be based upon them.

Cases of malignant endocarditis are generally diagnosticated as typhoid fever, and especially when no focus of suppuration or necrosis can be discovered, because the general appearance of the patient having endocarditis resembles, to a great extent, that of a patient suffering with typhoid fever. It is therefore very easy to confound a malignant endocarditis with typhoid fever. We should be careful in our observations and conclusions when we make a diagnosis of typhoid fever, bearing in mind that malignant endocarditis may simulate it, paying especial attention to the temperature curve, for it has been demonstrated that in malignant endocarditis the typical rhythmical exacerbations of typhoid fever are absent.

The disease has been mistaken for acute yellow atrophy of the liver. This should happen very rarely, as the symptoms of the latter will in most cases of that disease be too pronounced to allow of this error in diagnosis. It has sometimes been diagnosticated as intermittent or malarial fever on account of the chills and irregular temperature with intermissions and remissions. An examination of the blood for the *plasmodium malariae* will very soon settle this point. It has also been diagnosticated as acute miliary tuberculosis and as cerebral or cerebro-spinal meningitis. Clinically it may bear a resemblance to phthisis, but an examination of the sputa for the tubercle bacillus will determine the point. From the varieties of meningitis it may not be so easy to differentiate malignant endocarditis, and the examination of the patient for special symptoms of the meningeal inflammations should be made with great discrimination.

Before coming to a conclusion a careful examination for an infectious process or nidus should be undertaken. In a certain proportion of cases the disease has taken its origin of infection from some trivial injury, as a scratch of a pin, the rubbing of a shoe, the pulling off of a hangnail and the like.

In view of the difficulty experienced in establishing a diagnosis of malignant endocarditis, inoculation experiments upon rabbits and guinea-pigs, as has been done by Josseraud and Roux (*Lyons' Medical*, September 6, 1890), for the purpose of making

a positive diagnosis during the life of the patient, may be of value and are worthy of more extensive use.

If a patient exhibit no sign of previous valvular disease, but is the subject of a slight blowing systolic murmur with its intensity at the apex disassociated with definite rheumatic symptoms or other possible causes of such a murmur, but accompanied by grave signs of constitutional disease and prostration, the presence of malignant endocarditis is very probable. Finally, if in a given case, petechial hemorrhages be present with febrile action, and if hemorrhagic measles, hemorrhagic variola, epidemic cerebro-spinal meningitis and typhus fever, all of which have been mistaken for malignant endocarditis, can be excluded, then the case is in all probability one of malignant endocarditis.

The disease has been invariably fatal in all cases hitherto positively recognized. It seems that the records do not bear a single case which has recovered. Once the diagnosis shall have been positively made the prognosis will be very unfavorable.

The treatment of the disease will embrace the management of the local source of infection as well as that of the constitutional infection.

In the pyæmic and septic cases the measures appropriate thereto will be indicated. The diet should be regulated and should consist of easily digested and highly nutritious substances. The excessive activity of the heart should be diminished by cardiac sedatives. The fever should be reduced by the employment of the Brand cold bath, cold affusions and the cold pack; ice bags are of service to allay the headache and to lessen cerebral excitement. As parasiticides the mineral acids, quinine, salicylic acid can be given. In order to avoid gastric disturbance and weakening of the heart's action digitalis should be administered with great caution. To keep up a flagging heart glonoin strychnine, camphor, the double salicylate of caffeine, and musk are to be recommended.

The bichloride of mercury and phenic acid internally have been commended as germicidal, as have also the inhalations of oxygen and ozone.

Notwithstanding all the energetic treatment, these unfortunate patients seem doomed to die, and in view of the futility of all former kinds of treatment, I think we should turn to the new serum therapy of to-day for a remedy in this dire affliction. In these cases I would recommend, and would not hesitate to em-

ploy, the streptococcus antitoxin serum. As the older treatment has failed so signally, this streptococcus antitoxin treatment can do no less, and it is to be hoped that it will do a great deal more.

161 WEST FIFTY-SEVENTH STREET.

DISCUSSION.

DR. W. P. NORTHRUP.—I wish to call attention to a case at the New York Foundling Asylum. It was seen by Dr. J. Lewis Smith and Dr. O'Dwyer. This is the only case of endocarditis in a young child, which has appeared at the Foundling Hospital for many years. Several times cases have been seen which gave some of the symptoms of that condition, but upon autopsy, endocarditis was not present. It must be an exceedingly rare disease under four or five years.

DR. L. EMMETT HOLT.—In a series of 242 autopsies made upon cases of congenital cardiac disease there were four which died of malignant endocarditis. Three of these had lived to adult life. One was but four years of age. This child had malignant endocarditis, engrafted on cardiac disease. I may say in confirmation of Dr. Northrup's remarks in regard to the very great infrequency of endocarditis in young children, that in a record of 1,000 autopsies upon children under three years of age, not a single example was seen.

DR. WM. OSLER.—While simple endocarditis is so extremely common in children, the ulcerative form is rare. Infants are not nearly so frequently attacked. It is rather remarkable, considering the common association of endocarditis with the acute fevers and rheumatism in children, that so few cases assume the graver type.

GANGRENE OF THE LUNGS COMPLICATING TYPHOID FEVER.

BY GEORGE N. ACKER, M.D.,

Physician to the Children's Hospital, Washington, D.C.

C.K., male; white; aged eight years. Entered the Children's Hospital, December 23, 1895.

The family history was good. The child had always enjoyed good health, and had never had any of the usual diseases of childhood. For a week previous to his admission into the Hospital, he had been complaining of headache, pains in the abdomen, and loose bowels. He was delirious at times, and had a continued high fever.

Condition on admission. — The child appears to be well nourished, but somewhat anæmic. There are several rose spots on the abdomen. The tongue is coated in the centre, and red at the edges. The appetite is good and bowels are loose. There are some fine râles over both lungs. Pulse 144; temperature 104.6; respiration 60. He was so nervous and restless that it was necessary to put on a strait-jacket in order to keep him in bed. He was slightly delirious, and this increased as night came on.

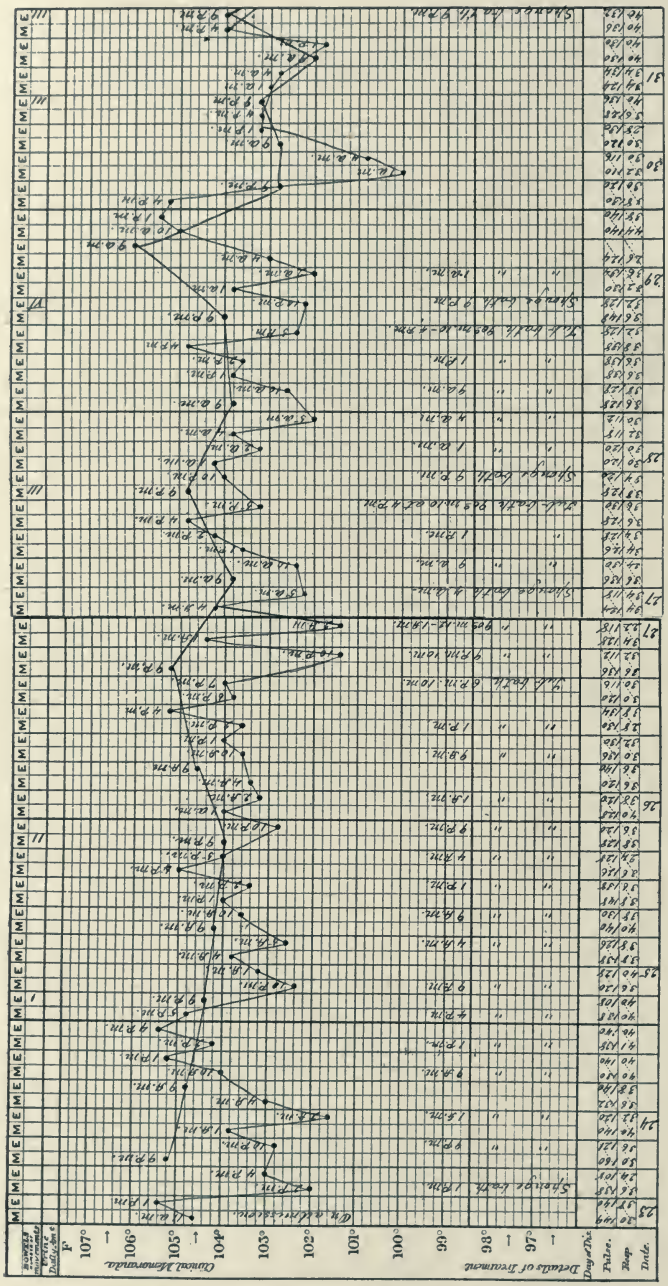
He was placed on a liquid diet, and sponge baths were ordered for the fever.

December 24. He has been very delirious, but the baths have had a quieting effect. He became so weak that whiskey was ordered to be given every three hours.

December 25. He was very restless during the night. The delirium is muttering in character with carphologia. He does not take milk well.

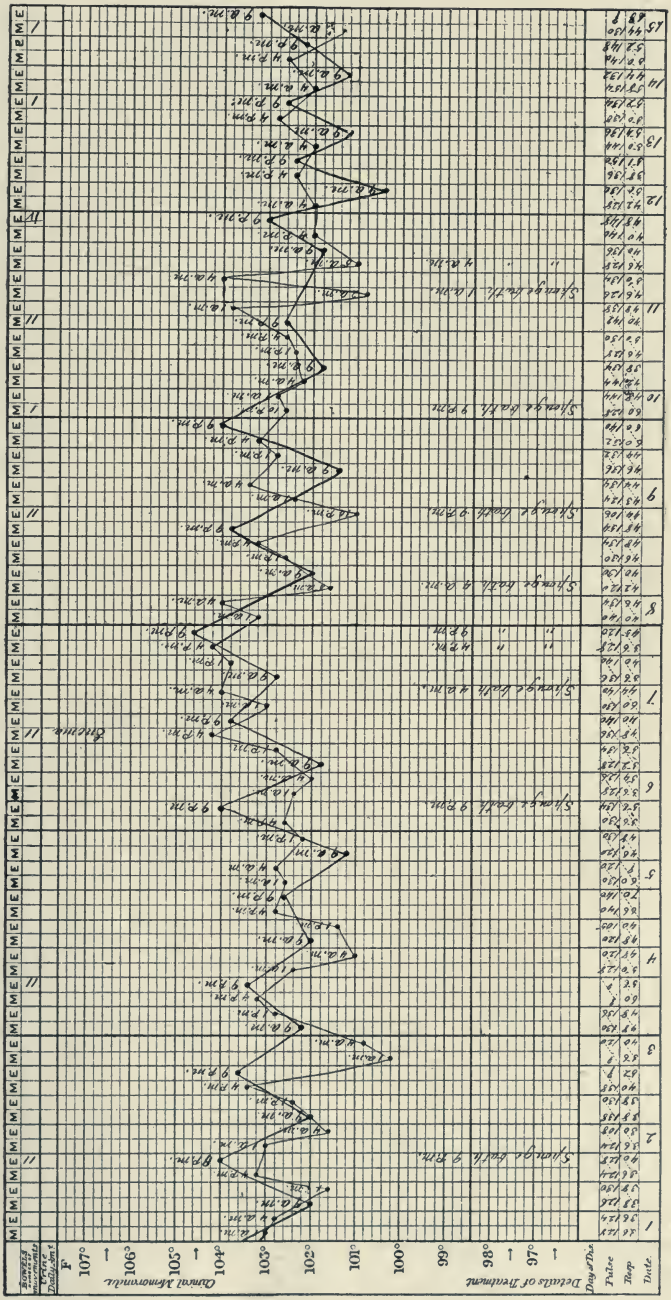
December 26. The boy has a frequent cough. He has a twitching of nearly all of the muscles, and this becomes more pronounced if any one disturbs him. As the sponge baths did not have any effect to-day, tub baths were substituted. These had a marked effect on the temperature, and delirium. Strychnine sulphate (gr. $\frac{1}{200}$) every four hours was ordered.

December 27. The muttering delirium continues, with



TEMPERATURE RANGE.

Gangrene of the Lungs Complicating Typhoid Fever.



TEMPERATURE RANGE.

Gangrene of the Lungs Complicating Typhoid Fever (Continued).

occasional jactitations. The slightest disturbance causes these manifestations. This makes it difficult to feed or to examine him. The child is continually picking at the air, and throwing off the covers. The pulse and respiration are good. The cervical glands are enlarged. The abdomen became very tympanitic, but turpentine stupes being applied, this was relieved. Tincture of hyoscyamus (m. xxv.) and sodium bromide (gr. v.) were given at night for the restlessness.

December 28. The cough continues frequent. There are many mucous râles in both lungs. He did not take nourishment well to-day, and he appeared to have some difficulty in swallowing. He is in an irritable state, and refuses to open the eyes. When in the baths to-day he became cyanosed, and the pulse was weak. Tincture of digitalis (m. v.) every four hours was given and all other medicine was stopped.

A suppository containing sulphate of codeine (gr. $\frac{1}{8}$); extract of belladonna (gr. $\frac{1}{6}$) and extract of hyoscyamus (gr. $\frac{1}{4}$) was given for the very nervous condition, and this had a good effect.

December 29. During the bath this morning the pulse and respiration became weak and rapid. The typhoid state is very pronounced with sordes on lips and teeth, muttering delirium, and carphologia. It was necessary to resort to nutritive enemata to-day, as the child could not swallow. There are numerous small and large mucous râles in both lungs. An ice cap was applied to the head and ice water cloths to the abdomen. This had a very beneficial effect, as he slept well and was less nervous.

December 30. The cough is frequent, but the air enters the lungs better, and he appears to be improved. He can take some nourishment by the mouth.

December 31. Very restless again. The ice water application renewed.

January 1, 1896. Both of the parotid glands are very much enlarged, and appear to be very painful. He is unable to swallow. Poultices were ordered to be applied to the glands. The breath is very offensive. There is dullness on the right side of the chest anteriorly, and both sides posteriorly. There are numerous fine mucous râles in both lungs posteriorly. The breathing is diminished in the right lung anteriorly.

January 2. The glands are more swollen and tender to the

touch. During the night the patient became so restless that hyoscine hydrobromate (gr. $\frac{1}{200}$) was administered hypodermatically, and an ice cap was ordered. The breath is extremely fetid, and he coughs more than usual. Sulphide of calcium (gr. ii.) every two hours was given.

He has been having a series of minor convulsions during the latter part of the afternoon. At one time the convulsions were quite severe, and he became cyanosed.

January 3. The pulse is weak, and the respirations are becoming rapid and irregular. Has a frequent weak cough. He is unconscious. The head and face are much swollen.

January 4. The breath is very offensive. The patient breathes with great difficulty using the auxiliary muscles of respiration, and extending the head. The calcium sulphide stopped. Incisions were made in the glands to-day, and about a teaspoonful of pus was removed from each of them. He reacted well from the operation; the breathing soon became better, and he was able to take food by the mouth. Later in the day the respiration became shallow and irregular, and deglutition difficult. He has an incessant cough.

January 5. The child is less nervous and takes nourishment well. The glands are smaller, and the breathing easier. The swollen appearance of the face and head is less marked. A bright papuler eruption appeared on the chest, and spread rapidly to the abdomen and lower extremities.

January 6. The respirations are improved in character, but the cough is frequent, and appears to exhaust the patient. The pulse is irregular and weak at times. The rash covers the entire body. Slept all day. Had a good stool to-day from an enema.

January 7. He is nauseated when he takes food and swallows with difficulty.

January 8. The child lies in a stupor. The lungs do not expand well. The eruption is less marked. The swelling of the parotid glands has disappeared.

January 9. The child takes food better.

January 10. The eruption has disappeared. He vomited the milk to-day, and was very restless.

January 11. He did not sleep at night. He understands at times when spoken to. The breath continues very offensive.

January 12. Yesterday and to-day he vomited food oc-

casionally. The treatment for some days has been stimulants with digitalis and strychnine.

January 13. He understands when aroused, but the hebetude is marked. The cough is the same. The heart sounds are good. There is dullness over both lungs posteriorly with bronchial breathing.

January 14. The condition of the patient is about the same with frequent cough and restlessness.

January 15. He was very restless during the night. Suddenly about 8 A.M. he became cyanotic with heavy breathing and a weak small pulse. He gradually grew worse, and died at 9:45 A.M.

Post mortem examination.—The body was poorly nourished; little adipose tissue present. The heart was normal.

When the right chest was opened the pleura were found adherent, and about a pint of pus escaped when the lung was removed. Both of the lungs had a large gangrenous abscess posteriorly containing dark blood clots. There were, also, several small similar abscesses. The tissue around the abscesses was extensively consolidated. The pleura was covered with membranous lymph. The gangrenous odor was marked. The vertebral column and ribs were eroded in spots.

The spleen and mesentery glands were enlarged. The liver was normal in size.

The intestines presented marked evidences of typhoid fever. Many of the ulcers of Pryer's patches had healed and a few were still in the process of repair.

913 SIXTEENTH STREET.

A CASE OF ENDOTHELIOMA OF THE CEREBRAL MEMBRANES WITH JACKSONIAN EPILEPSY AND WASTING OF THE PARALYZED MUSCLES.

BY FREDERICK A. PACKARD, M.D.,

Visiting Physician to the Children's and Philadelphia Hospitals; and to the Out-patient Department of the Pennsylvania Hospital. Instructor in Physical Diagnosis, University of Pennsylvania.
Philadelphia.

The following case is reported on account of the nature of the tumor, the localizing symptoms, and the muscular atrophy present in the paralyzed muscles:

May R., white, aged fourteen years, was admitted to the Children's Hospital on July 11, 1895, having been brought from her home in Delaware on account of what was supposed by her family to be some form of St. Vitus' dance.

The family history was negative. She had been ordinarily healthy, having had trouble with the bowels during dentition and chicken-pox at the age of six years. She had had no other diseases and had not received any injury.

Ever since the preceding summer she had been irritable, dull and forgetful, and had complained more or less of headache. On February 12, 1895, she was sitting in a chair, when she suddenly stopped talking and working and fell off the chair to the left side. She at once began to have twitching of the left arm and leg, which lasted for about two minutes. There was no loss of consciousness. She recovered from this attack and was in her usual condition for a few days, when a second convulsive seizure, limited to the arm, leg and neck of the left side, occurred. Ever since that time (five months before her admission to hospital) she had frequent convulsions, sometimes several occurring in one day, sometimes a week passing without a seizure. They usually were preceded by a vague aura that warned her of their approach, were always upon the left side of the body, usually lasted but a few minutes, and were not accompanied by loss of consciousness. From the first she had much pain in the neck and left shoulder during the convulsions, but for a month prior to her admission had complained of constant pain in those regions. In the course of time (but exactly at what time could not be definitely determined) the convulsions gradually assumed the character described by her mother as follows: The truncal muscles ceased to be involved in the convulsive movement; the left heel touched and rebounded from the floor; the left forearm was flexed and extended, the hand tapping the abdomen. The left eyelid, w hich

at first participated in the seizures, gradually ceased to twitch. During the convulsions she never bit her tongue or soiled the clothing. Five weeks before admission she suddenly became blind and remained so for a half day, complained of intense headache, seemed very "nervous," and yet apparently was somewhat stuporous. On the next morning she had regained some vision, but this had gradually become again diminished until July 6th, when she again became totally blind, and has so remained.

Ever since the first symptoms of illness (a year before her admission) her mental power had steadily failed and she had complained more and more of headache.

Six weeks before admission she began to have vomiting at frequent intervals, independently of taking nourishment; while a month before admission there had been almost constant spitting of saliva, which continued for two weeks.

The convulsions became less severe and of shorter duration about a month before her admission, and during that time she had been confined to bed.

In regard to the loss of power in the left side nothing could be learned from the mother, as she did not know that this side was useless even when she brought the girl to the hospital.

The only other facts of interest in the history are that her appetite was good, although she frequently at once vomited ingesta, that the bowels were usually constipated, and that she urinated at very long intervals. There had at no time been any discharge from the ears.

Examination on admission showed that she was a thin, almost emaciated, girl of average height. The face was thin and wan, the left side seeming to droop slightly and to be less mobile on grimace. The pupils were widely and equally dilated and there was slight nystagmus. The teeth were covered with sordes, the gums bleeding easily, the tongue protruded slightly to the left and thickly coated with yellowish white fur, the mouth filled with thick, sticky saliva. The temporal region on the right side was swollen and reddened, apparently the seat of former vesication. Just back of the vertex there was a conical tumor, sessile, hard, evidently firmly connected to the calvarium, and measuring one inch in diameter at its base. There was quite marked rigidity of the nucha and it was noted that on raising the head to test for rigidity the pupils seemed to dilate even more widely than when quiet.

The decubitus was dorsal, with slight inclination toward the left side. The respirations were very infrequent, the pulse slow and feeble. Examination of the heart, lungs, liver, spleen and abdomen were negative. Over the thighs and legs were several pigmented spots of about an inch in diameter and over the whole lower portion of the back was a huge area extending from the lower ribs to the end of the sacrum and from one posterior ax-

illary line to the other, which was of a rather reddish color and was covered (as also to a less extent were those on the thighs and legs) by a rank growth of coarse brownish hair resembling the fur of a lower animal rather than that seen normally on any portion of the human body.

The left arm lies relaxed and motionless, the fingers being semi-flexed and the thumb in close apposition to the metacarpal bone of the index finger. The arm is distinctly more wasted than the rest of the body and much more so than could be attributed to lack of use. The wasting is most marked in the shoulder region, in the thenar and hypothenar eminences, and in the interossei. In fact, the arm resembles precisely that of an advanced case of idiopathic muscular atrophy or that due to chronic anterior polio-myelitis. All of the deep reflexes were absent, muscular irritability slightly increased. To coarse tests there was no sensory disturbance, but the child's condition was so bad and she was already so exhausted by the examination that exact estimations as to quantitative or qualitative loss was out of the question. The right arm was wasted to a degree commensurate with the general emaciation, but showed none of the atrophic muscular appearance so marked on the left side. The grasp was extremely feeble, but there was no sign of local weakness beyond that explainable by her general physical condition. The deep reflexes could not be elicited, the muscular irritability was slightly increased.

The left leg was absolutely powerless, the right moved as vigorously as would be expected in her condition. The deep reflexes were absent on both sides. There was no evident difference in the musculature of the two legs.

Dr. Geo. E. de Schweinitz kindly examined the eyes and reported as follows: "Total blindness. Catarrhal conjunctivitis and œdema of lids. Left palpebral fissure narrower than right. Double optic neuritis, swelling of disc on right side subsiding (+ 2 D) and temporal edge beginning to appear, nasal margins still hidden. Swelling on left side much higher (+ 5 D) and all margins hidden by inflammation. No hemorrhages. Slight nystagmic movements of eyeballs, but no deviation of visual axes. Pupils dilated *ad maximum* and irresponsive to light."

Dr. B. A. Randall kindly examined her ears and reported absence of any lesion.

The urine had to be obtained by catheter and showed no abnormal constituents. Her general condition was so wretched, probably on account of her long journey, that she required quite active stimulation, and save for this and carefully selected nutritious food, was given no treatment.

During the night after admission, she had an attack which was described by the nurse as a shaking or trembling of the whole body. During July 12th and 13th she had four attacks, lasting from two to three minutes, not accompanied by loss of

consciousness, but on the contrary by excitement and faint cries for help and relief. The attacks were not seen by myself but were described by the resident physician, Dr. Max Blieden, as follows: "The movements began sometimes in the left hip, at other times in the left shoulder; but in whichever member they began, in the course of a few seconds to a half minute, the movements soon involved the other member of the left side. They consisted of abductions and adductions of the arm, flexion and extension of the left leg, in their character and time resembling those that would be produced by a slowly interrupted faradic current. After these movements had lasted for about a minute, the left forearm and left leg were observed to be flexed and extended. The movements then gradually became slower and died away."

Her other symptoms may be briefly enumerated. The temperature never varied beyond the line of 98° and 99°. The pulse was constantly feeble and usually slow, but never below 60 to the minute. The respirations were slow except toward the end of her attack, being about 10 to the minute. At intervals they took on a typical Cheyne-Stokes character. She usually lay perfectly quiet and in a semi-stuporous state, but could be aroused. The urine had to be withdrawn by catheter for the first twenty-four hours, but was later passed in bed apparently unconsciously.

She never rallied from the effects of her removal, and during her stay in the hospital she received practically no treatment save that required to stave off an apparently constantly imminent death. She never reached such a condition that operation could be thought of, especially in view of the large extent of growth indicated by the signal symptoms. She died on July 14th, three days after admission, from gradually increasing asthenia, death coming through apparently simultaneous cardiac and respiratory failure.

Permission for general autopsy was refused, but examination of the brain was allowed. In moving the calvarium it was found that beneath the site of the superficial tumor noted during life the dura and bone were solidly united together and the latter was also welded to the parts below, requiring separation with the knife. It was seen that the dural growth was a continuation of the tumor to be described below, and that the tumor of the bone was evidently simply an extension of the process through the vault. The bone substance was friable and its cut surface showed alternating spicules of bone and softer tumor tissue. The vessels over the convexity were filled with blood, especially on the right side. The brain was removed and at once placed in Müller's fluid for more detailed study.

Except for the tumor mass to be described below, the general appearance of the brain did not differ from the normal. The base appeared to be perfectly normal and there was no evidence of transmitted pressure. On the under surface of the left tem-

poro-sphenoidal lobe, and on the lower surface of the left frontal lobe, close to the origin of the fissure of Sylvius, and at the apex of the right temporo-sphenoidal lobe there were three brownish areas which, before immersion in Müller's fluid, had a rather angeiomatous appearance but were not elevated above the surface and showed no marked increase in resistance.

The consistence of the brain itself appeared about normal. On the convexity nothing abnormal was found upon the left side. Lying immediately beneath the dura mater in the parietal and posterior portion of the frontal region on the right side there is a mass 10 c. m. long, extending outward 5 c. m. from the median line and 4.5 c. m. downward on the median surface in the great longitudinal fissure. The tumor looks as though it were laid on the cerebral substance, there being a distinct overlapping edge resembling the edge of a plaster of Paris mould. The mass extends from a point 3 c. m. anterior to the parieto-occipital fissure forward to about the level of the bifurcation of the arms of the fissure of Sylvius. It does not encroach upon the lateral surface of the hemisphere. It directly covers almost the entire upper portion of the parietal lobe bordering on the longitudinal fissure and also a very considerable portion of the frontal lobe in its upper part. It involves the upper portion of the Rolandic fissure and the ascending parietal and frontal convolutions. It does not reach the supra-marginal or angular gryri.

The tumor was firm on section, although smooth and uniform. The neighboring brain-substance was softened and blood-stained. It appeared as though the mass had sprung from the pia-arachnoid and had pressed upon without infiltrating the cortex.

The cerebellum, pons and medulla were unaltered in shape or consistence.

Sections of the tumor, kindly made for me by Dr. J. Dutton Steele, present the typical variegated appearance of an endothelioma.

Microscopic examination of sections of pons, medulla, and upper portion of cord (that removed with the brain being all that could be obtained by section through the foramen magnum) shows absolutely no sign of descending degeneration when stained by Weigert's method, nor do the cells in the anterior horns of the portion of spinal cord show any abnormal characters.

The essential features of the case may be summed up as follows: Without known predisposing or exciting cause there had been mental disturbance of low grade with frequent cephalalgia for one year. Five months before death a sudden left-sided convulsion. Frequent left-sided convulsions continued even up to death. Blindness came on five weeks before admission but lessened until five days before admission, when sight was again lost. On admission there was found, beside choking of the discs, absolute blindness, absolute palsy of left arm and leg, and distinct atrophy of the paralyzed arm. During her stay in the hos-

pital she had numerous epileptiform convulsions of Jacksonian type occurring in the paralyzed parts. At autopsy beside the endothelioma with its physical evidences of pressure upon the motor cortex of the right brain there was found no lesion of the central nervous system in so far as it could be examined.

The author looks upon this as one of the rare cases of cerebral muscular atrophy from tumor. Several objections to the theory at once occur to the mind. In the first place the occurrence of muscular atrophy from cortical disease is opposed to our ordinary ideas in regard to the situation of the trophic centres for the muscles of the periphery which are supposed to be located solely in the multipolar cells of the anterior cornua of the spinal cord. The apparent atrophy might be claimed to be the result of disproportion between the two arms from lack of development in the palsied member as compared with the corresponding part of the opposite side. The atrophy might be thought to be due to simple atrophy from lack of use.

The first objection will be considered below in discussing the various forms of atrophy occurring from cerebral disease and the theories that have been held in regard to their immediate cause.

It is much regretted that, owing to pressure of work from the crowded condition of the wards with serious cases during the hot weather present through this child's hospital life, no measurements of the arms or legs were made nor any tests performed in regard to the electrical conditions present. The author realizes that absence of the latter is a serious omission in the study of the case, and impairs its usefulness.

That it was a true atrophy from central disease cannot be doubted in view of the great diminution in size and of the marked atrophy of the deltoid, thenar and hypothenar eminences and interossei typical of the usual atrophy from multipolar cell disease or idiopathic (muscular) atrophy. The arm lay absolutely flaccid, the normal contour of the shoulder was entirely gone; the hand had the peculiar simian shape, the thenar and hypothenar eminences being flattened, the interossei wasted, the thumb lying parallel with the radial border of the second metacarpal and index finger. The changes were not such as would indicate simple lack of growth, as is seen so commonly in the cerebral palsies of children; and, indeed, it is hardly conceivable that so great a difference in the two arms as

was present in this case could be accomplished by the well arm in the short space of five months. Diminution in size from relative ack of growth would also not show the peculiar distribution ot the more marked wasting seen in this case. Bastian* indeed says that arrested or retarded growth occurs between birth and the third or fourth year, much below the age of the subject of this paper. Atrophy from disuse is a cause that might be suggested as present in this case. This could not explain the atrophy, inasmuch as this arm was being quite regularly and violently exercised in the convulsions that were so marked a feature—an argument that was used in the same way by Senator† in explaining his case of muscular atrophy following cerebral abscess. Babinski‡ says that atrophy from lack of use is slow, is never marked, and may be completely absent even if paralysis continues for a long time. Certainly in the present instance the whole appearance of the arm, as well as attentive observation of the various parts of the arm and hand, decided against atrophy from disuse as is seen after injury or in other conditions where the member has been long at absolute rest. If these facts are correct it follows that, in view of the healthy condition of the motor path in the pons, medulla and upper fragment of the cord, the atrophy must have been due to either the lesion found in the brain, to a localized polio-myelitis in the cervical region of the cord, to a neuritis of the nerves of the arm, or to idiopathic changes within the muscles themselves. The existence of the latter condition with the brain tumor would be a curious coincidence; neuritis is excluded by the absence of sensory changes; while it is hardly likely that a limited polio-myeletis of the cervical region of the cord would be apt to occur simultaneously with the brain lesion. The possibility of the occurrence of these causes for the atrophy cannot be excluded here, owing to the limitations imposed by the family in granting permission for the autopsy.

There are now reported a large number of cases of atrophy resulting directly or indirectly from cerebral disease, and while by their number these establish the possibility of such an occurrence they cannot yet lead to any practically useful con-

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* *London Lancet*, September 19, 1874, p. 405.

† *Berlin klin. Wochenschr.*, January 27, 1879, et seq.

‡ *Archives de Neurologie*, July, 1886, p. 591.

clusions. While Dana* says, in discussing apoplectic hemiplegia, that the affected muscles do not waste, and that the muscles, though but little used, do not become smaller to any notable extent, it is rather astonishing to see the number of cases of such atrophy reported, especially by Charcot and his followers. In the discussion upon Putzel's case† at the New York Academy of Medicine, cerebral muscular atrophy was considered as an extremely rare phenomenon by all who spoke. Bastian‡ says that an early and special wasting of the muscles is uncommon, and that only a few cases were at that time on record. Pitres,‖ in discussing his case of muscular atrophy following descending degeneration due to a focus of cerebral softening, says that it is very exceptional. C. Eisenlohr§ says that slight atrophy of the small muscles of the hand is not rare in ordinary hemiplegia produced by hemorrhage or softening, but that more marked or especially widespread atrophy is rare. Steiner¶ says, in his most complete article on muscular atrophy in cerebral hemiplegia, that there occurs a sufficient number of cases of cerebral hemiplegia with muscular atrophy without descending degeneration to make its existence certainty, although it is a rare condition.

In cases of tumor of the brain atrophy would appear to be quite rare, inasmuch as Starr[a] says that there is no atrophy in the paralyzed muscles, but merely a slight wasting from disuse, in cases of this character, and Bremer and Carson[b] were able to find but five cases in medical literature, their own case making the sixth.

The muscular atrophy of cerebral origin can be divided into early and late, in regard to its time of appearance after the cerebral disturbance. The case under consideration, while it did not occur (to our own personal knowledge) at so early a date as did some reported—*e.g.*, Borgherini's[c] case of supposed hemorrhage

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* "American Text-Book of Nervous Diseases," p. 458.

† *N. Y. Medical Record*, 1882, Vol. I., p. 609.

‡ *Loc. cit.*

‖ *Archives de physiologie normale et pathologique.* Second series. T. iii., p. 657, 1876.

§ *Neurologische Centralblatt*, January 1, 1890.

¶ *Deutsche Zeitschrift f. Nervenheilk.* Bd. iii., Hft. 4 and 5.

a "American Text-Book of Nervous Diseases," p. 490.

b American Journal of the Medical Sciences, February, 1895, p. 133.

c Deutsches Archives f. klin. Med. Bd. xlv., p. 371, 1889.

in the region of the optic thalamus with decided atrophy of the palsied arm in two days—yet, inasmuch as the first sign of trouble with the left arm occurred only five months before atrophy in marked degree was observed by us, it is reasonable to class this with the early rather than the late hemiplegic muscular atrophies.

The cerebral atrophies may be classified anatomically into four classes: hysterical without anatomical lesion at any part, organic with brain lesion alone, organic with brain lesion and descending degeneration, organic with brain lesion, descending degeneration and involvement of the cells of the anterior horn of the spinal cord. Steiner's table of classification is as follows:

	BRAIN.	PYRAMIDAL TRACTS.	ANTERIOR HORNS.	FORM.
1.	Visible Alteration.	Visible Alteration.	Visible Alteration.	Spinal.
2.	Visible Alteration.	Visible Alteration.	Invisible Alteration.	Cerebral.
3.	Visible Alteration.	Invisible Alteration.	Invisible Alteration.	Cerebral.
4.	Invisible Alteration.	Invisible Alteration.	Invisible Alteration.	Hysterical.

In this classification our case would come under the third category. In this respect it agrees with the cases of Rott and Mouratoff,* Quincke,† and Bremer and Carson.‡ In Pel's case the condition of the spinal cord is not stated, while in Patella's case, as given in Steiner's table of cases, there is no mention of the presence or absence of descending degeneration. Burresi's case, as given by Quincke, had sclerotic foci in pons and medulla. Quincke's third personal case did not come to autopsy, having been improved by treatment, the diagnosis of gumma in the motor region of the cortex having been made. While in this case autopsy was not made, for obvious reasons, it is presumable that there could not have been very extensive anterior horn disease. Quincke's second case had not so favorable a termination, and, while marked atrophy was present in the left arm and hand, there was no central nervous lesion save for the presence of a glioma involving the central convolution, the paracentral lobule and the foot of the second frontal convolution on the right side without descending degeneration.

Of Steiner's eighteen cases, thirteen had atrophy of the left

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*Contribution à l'étude de la Pathologie des Hémipphéres Cérébraux, Moscow, 1890, as given in review by J. Roubinovitch in *Archives de Neurologie*, 1891, Vol. xxi., p. 296.

† *Deutsches Archives f. klin. Med.*, 1888, Vol. xlii., p. 492.

‡ *Loc. cit.*

extremity, the side upon which atrophy was present in my case.

Atrophy from cerebral disease may pathologically vary from the invisible disturbance of hysteria to that produced by atrophy of the anterior horn cells from transgression of the degenerative process from the lateral columns. While for the teaching of students it is advisable to, so to speak, diagrammatically divide the nervous system into two portions—that from the brain cortex to the ganglion cells of the spinal cord, and that from the latter to the intra-muscular nerve endings—and to state that trophic influences are exerted over any particular portion by the multipolar cell next above, yet there are now a sufficient number of cases reported of trophic lesions in the muscles following disease strictly limited to the brain, to cause some extended inquiry into the strict propriety of this teaching.

Several theories have been proposed to account for these rather unusual, but still not unheard of cases of muscular atrophy after cerebral disease. The easiest, and at first sight most attractive theory, is that which holds that in the motor cortex there are cells having a trophic influence over the muscles. A fatal argument to this theory is that no one portion of the brain can be mentioned, lesion of which necessarily or even usually is associated with atrophy of muscles; although it would seem from the reported cases that the motor region of the cortex, or the motor paths therefrom, are always the seat of trouble. Still, Steiner's statement that "purely statistically trophic brain-centres do not exist" must be accepted. Charcot's explanation of the atrophy observed in old cases of hemiplegia—invasion of the anterior horn cells by the sclerotic process from the lateral columns—will, of course, not explain the quite numerous cases wherein no descending degeneration is found, while according to Joffroy and Achard * the sclerosed area is always separated from the anterior horns by a band of normal tissue.

Quincke and numerous others adopt the theory that the atrophy from brain lesion, without descending degeneration or lesion of the ganglion cells in the anterior horns of the spinal cord, is due to the withdrawal of a trophic influence which the cortical cells exert upon the motor part of the spinal cord, and therefore indirectly upon the peripheral nerves and muscles, and

* *Archives de Médicine Expérimentale et d'Anatomie Pathologique*, 1891 iii., p. 780, *et seq.*

(to quote Quincke), "that nutritional disturbances of the latter can even appear if the deficiency of the cortical influence has produced functional, but not any recognizable anatomical damage of the paths of the spinal cord." He thinks it possible that the trophic fibres may run in some path removed from that of the motor fibres, and in that way explains cases of palsy from motor path lesions (as in the internal capsule), which remain for a long time without atrophy occurring. Borgherini suggests that these fibres from theoretical trophic cortical centres may go through the sensory tract; but here again, as lesions in any part of the brain may be found without atrophy, the path cannot be considered as determined.

That some trophic influence is exerted by the cortex upon the peripheral portions of the nervo-muscular structures seems to be rather confirmed by the existence of atrophy in hysterical palsy; but our knowledge of hysteria is so vague that it is not safe to base any conclusions upon analogy with that condition. This much can, however, be said, that if there be such a trophic influence in hysteria, the atrophy is due to dynamic changes in the pyramidal or other tracts of the spinal cord, and in the anterior horn cells, inasmuch as palsy and atrophy usually disappear together.

Rott and Mouratoff attribute the atrophy to ischæmia of the parts from narrowing of the blood-vessels thereof, and argue that each psycho-motor impulse from the cerebral cortex is accompanied by a vaso-motor impulse having for its object the carrying of a certain quantity of blood to the corresponding muscles. All that can at present be said, therefore, in regard to these cases of muscular atrophy occurring in the course of cerebral lesions that are not followed by spinal cord changes, is that there seems to be some trophic connection between the motor cortex or paths therefrom with the peripheral muscles, either directly, or by way of the ganglion cells of the anterior horns, and that this trophic influence may be withdrawn without anatomical changes in the pyramidal tracts or ganglionic cells of the cord that are visible with our present means of observation; that, granting the existence of such trophic functions in the cortex, the path of the trophic fibres is not known.

The author would call attention to the fact that, with the six cases mentioned by Bremer and Carson and with one of Quincke's not included in their paper, the present case raises the number

of reported cases of brain tumor with muscular atrophy to eight. It is also a fact to be noted that in this case the lesion concerned the right side of the brain, as was the case in 13 out of the 18 cases of muscular atrophy following various cerebral lesions that were tabulated by Steiner.

110 SOUTH EIGHTEENTH STREET.

MULTIPLE PAPILLOMATA OF THE LARYNX, IN A CHILD OF THIRTEEN MONTHS; TRACHEOTOMY; IMPROVEMENT; DEATH FIFTY DAYS AFTERWARDS FROM PHARYNGEAL AND TONSILLAR DIPHTHERIA.

BY IRVING M. SNOW, M.D.,

Clinical Professor Diseases of Children, University of Buffalo; Physician to Buffalo Fresh Air Mission Hospital.

The patient was a boy of thirteen months. During the first six months of life, he was in perfect health, able to phonate and make the inarticulate sounds peculiar to infancy. In May of 1895, the child was ill one week with a bronchitis, complicated by a facial eczema. After this illness there was complete and permanent aphonia. During the summer he suffered from a mild diarrhœa, but excepting for the loss of voice, passed through the fall and early winter in good condition.

December 4, 1895, I first saw the child, who was brought to me on account of its persistent loss of voice, which the parents had hitherto hoped would disappear. He was a strong, well developed boy, able to walk, sleep and eat well. He was possessed of five teeth, was suckled and fed and able to nurse continuously one-half hour without becoming short of breath.

There was complete aphonia. In crying, the child apparently went through a pantomine of grief, making no audible sound. It had habitually a loud, stridulous breathing. There was no cyanosis, no dyspnœa. During inspiration, there was recession of the lower ribs like a child with membranous croup. Heart sounds normal, apex in left fourth interspace in nipple line. Throughout both lungs, interiorly and posteriorly, there was hoarse breathing, with prolonged expiration.

On percussion, the chest seemed less resonant than normal at the bases, liver one-half inch below the ribs. The mother related that at night when she fed the child, it would often cough until it vomited. There was, however, nothing in the throat excepting perhaps a sensitive reflex.

From the history of the case, chronic aphonia, I diagnosed a laryngeal tumor, and Drs. Roswell Park and Renner were called in for operative treatment. It was impossible to make a laryngo-

scopic examination, but a growth protruding above the glottis could be felt with the finger.

December 13th. The child was sent to the Buffalo General Hospital and during the night had three dangerous attacks of dyspnœa, the first occurrence of this symptom.

December 14th. Tracheotomy was performed by Dr. Roswell Park. After the operation the child was greatly relieved of its dyspnœa and labored breathing. Recession of the lower ribs during inspiration was still observed. The patient remained in the hospital fifty days, enjoying better health than before the operation.

In February, 1896, a house epidemic of diphtheria occurred; five babies were attacked, three died. My patient was infected and succumbed in three days. The disease was tonsillar and pharyngeal; the membrane was thick and contained Klebs–Loeffler bacilli; antitoxin was used; the child died of heart failure.

(Borneman reports a nearly similar case in a boy of fourteen months, hoarse since the sixth week of life; the child began to suffer from dyspnœa. Its condition grew progressively worse, thyrotomy, larynx filled with papillomatous growths, mucosa, curetted, partial improvement, canula left in place. Two months afterward, the child died from diphtheria, the autopsy revealing extensive recurrence of the laryngeal papillomata.)

Autopsy.—Larynx examined. There was, curiously enough, no trace of inflammation or diphtheritic exudate in the larynx. Sir Morell Mackenzie states that in the presence of an epidemic of diphtheria a child with a laryngeal neoplasm is more likely to be attacked and less likely to recover. Five of the children in the hospital exposed contracted the disease in spite of preventive inoculations, two who had no laryngeal growth, died. Moreover the vulnerable region, the laryngeal mucosa, with my patient was free from diphtheritic disease. So there was nothing in this case to indicate any especial predisposition to diphtheria.

The larynx was the seat of multiple papillomata (an unusually typical specimen of the condition, uninjured by operation).

The growths were of the cauliflower, warty-type, and almost completely occluded the lumen of the larynx, extending above and below the rima glottidis. Looking from above it was impossible to see the true or false vocal cords. There were three

distinct masses, sessile in attachment, springing from the lateral and posterior portions of the mucous membrane.

Posterior tumor, irregularly oblong in shape extends from the free border of the inter-arytenoid ligament, downward nearly to the inferior border of the cricoid.

Tumor on the right attached to the false cord, fills the fossa of Morgagni and springs from the true cord and the mucosa beneath.

The growth on the left side, smallest in size, is attached to the true cord and the mucous membrane below.

It was evident that these extensive papillomata produced permanent stenosis from their nearly filling the interior of the larynx. To this permanent obstruction was added the occurrence of laryngismus so frequent in babies. The tumors were not sufficiently mobile or pedunculated to be caught between the vocal cords.

Etiology.—The origin of the growth was probably from a catarrhal tracheo-bronchitis at six months of age. There was no reason to suppose that the papillomata were congenital as until May, 1895, the baby was able to phonate perfectly. After the tracheo-bronchitis, the child became quickly aphonic. The case did not exhibit the usual slowly progressive symptoms, hoarseness, aphonia, dyspnœa, so that the tumor must at once have taken on a rapid growth.

These neoplasms are exceedingly dangerous in babies, on account of the liability of the subject to sudden suffocation from mechanical obstruction, to acute infectious processes, and to the peril of operative treatment. The endo-laryngeal removal is impossible, thyrotomy is hazardous; tracheotomy and intubation offering the most relief. The ultimate mortality from the condition in infancy is very great. In babies, the brassy cough and hoarse voice produced by laryngeal tumor may be ascribed to catarrhal laryngitis (croup) until death from acute asphaxia reveals the gravity of the lesion.

The case just described is one of the youngest on record, congenital growths excluded. Laryngologists of wide practice have reported a few laryngeal tumors in babies. Papillomata in infancy have curious characteristics exhibiting a great tendency to return after removal. They are capable of a very rapid growth and tissue infiltration like an acute infectious process (Gerhardt). Occasionally after a thyrotomy and through curetting of the

laryngeal mucosa, permanent cures are effected. Sometimes the laryngeal neoplasm has been coughed out in the paroxysms of pertussis. (Case of Dr. H. A. Johnson.)

There is no evidence to show that papillomata of the larynx in children undergo malignant degeneration.

476 FRANKLIN STREET.

DISCUSSION.

DR. W. P. NORTHRUP.—I should say that this is a rare lesion. As the larynx is filled with a mass of soft tissue I would suggest that the best treatment would be gentle, continuous pressure. This could be best applied by means of a tube. Such treatment, I believe, would cause considerable absorption.

DR. T. M. ROTCH.—Papillomata of the larynx is not so very rare in children.

DR. L. EMMETT HOLT.—The treatment of such cases is usually very unsatisfactory in young children. Broncho-pneumonia frequently follows operation. The incision is so near the seat of disease that it is difficult to prevent infection from extending downward. Intubation should be tried in cases like this. Although there is but slight probability of its being curative with a large tumor, it might be so if the papillomatous mass were favorably situated.

DR. A. CAILLÉ.—Laryngeal new growths are not so very uncommon in children. I know of two cases in New York at present in which intubation has been unsuccessfully tried with a view of getting polypoid growths to disappear by pressure. A number of cures are reported in which laryngotomy was performed and the growth removed. I believe with Dr. Holt that operative interference is frequently followed by an infection of the lungs in young children, but in older children the results are not so bad. Most surgeons hold that the splitting of the larynx and removal of the growth is the best procedure.

DR. A. SEIBERT.—It seems to me that if a tube is put into a larynx that gentle harmless pressure might easily become harmful pressure.

ORGANIC DISEASE OF THE HEART FOLLOWING SCARLET FEVER.

BY GEO. N. ACKER, M.D.,

Visiting Physician, Children's Hospital, Washington, D. C.

H. W., white, female, aged eight years, entered the Children's Hospital, January 10, 1895.

The mother is very nervous. The father is subject to rheumatism, and is a free drinker. The child has always been nervous. She had pertussis at three years of age, and scarlet fever when five years old. This was complicated by dropsy. Since then she has had pains in her back and legs at irregular intervals. She would have severe pains along the spine, with opisthotonus. When walking, would suddenly lose the use of the legs. She had occasionally choraic movements, with contracture of the hands and feet. She could not lie on the left side. With excitement the heart action became very violent. The child has lost weight lately, and has had night sweats.

Condition on admission.—The patient is anæmic and poorly nourished. Tongue coated. Breath fetid. Appetite poor, and bowels regular. Pulse weak, and circulation poor. Genito-urinary system normal. Has marked choraic manifestations. Ordered rest in bed, with moderate diet. A teaspoonful of the elixir of iron, quinine and strychnine three times daily; three drops of Fowler's solution three times daily, to be increased a drop daily, until ten drops are given at a dose.

January 26th. Marked improvement.

February 1st. On account of puffiness of the eyelids, the Fowler's solution was reduced to five drops three times daily.

March 15th. The choraic movements have disappeared. The child has improved very much. She is still anæmic. A soft, blowing murmur is heard over the base of the heart, extending to the right. The arsenic was stopped, and a dessertspoonful of phosphatic emulsion given three times daily.

March 24th. The impulse of the heart is to the left of the nipple.

April 6th. Strong action of heart for several days. The murmur continues over the base, and the other sounds are dull and muffled. Pulse 160, temperature 100°. She has some pains in the body and extremities. Other treatment stopped, and sodium

salicylate, five grains every three hours, ordered. For the next three days the patient had a daily range of temperature of 101° to 103°; pulse, 116; and respiration, 26.

April 11th. Temperature normal. The sodium salicylate stopped, and former treatment resumed.

May 1st. For some days the child has had a rise of temperature, 101° to 103°; pulse, 130 to 140; respiration, 26 to 40. Area of visible cardiac impulse is much increased. At the apex there is a double murmur, which becomes more marked toward the axilla. The other sounds are muffled and prolonged. The area of cardiac dullness is increased. There is broncho-vesicular breathing over the left lung anteriorly.

May 2d. Dullness upper part of left lung. Bronchial breathing at apex, and broncho-vesicular breathing at lower part of the left lung.

May 3d. The child is very weak. She cannot move with ease. Appetite poor and bowels regular. Tongue coated and pale. Has a frequent cough. Some râles on the right side. Left lung about the same condition. Ammonium carbonate, two grains, and fluid extract of digitalis, one-tenth minim, were given every three hours. Other treatment stopped.

May 8th. Has perspired freely for some days. Physical signs about the same, with sonorous râles posteriorly.

May 10th. Fine râles over both lungs. Bronchial breathing upper part of left lung with bronchopony. The past two days the temperature has been 102° to 104°; respiration, 42; pulse 140 and compressible.

May 11th. Marked dullness on left side, with bronchial breathing. Spartein sulphate (gr. $\frac{1}{10}$) every four hours. Other treatment stopped.

May 13th. Heart's action very rapid. Many râles both sides of chest. Fluid extract of digitalis (m. $\frac{1}{10}$) every four hours.

May 18th. Growing weaker. Liquid râles over both sides, posteriorly.

May 23d. Short broncho-vesicular breathing right lung with sonorous râles. Nitro-glycerine (gr. $\frac{1}{100}$) every three hours, spartein sulphate (gr. $\frac{1}{8}$) every three hours.

May 31st. Since the 18th, the temperature range has been from 100° to 101°; pulse, 140 to 160; respiration, 36 to 50. Perspires freely. Has slight choraic movements of fingers. Respiration diminished on the left side, and of a harsh character.

Broncho-vesicular breathing on the right. Heavy action of the heart. Purring thrill over the base of the heart. Apex beat three-quarters of an inch to the left, and one and a half inch below the nipple. Murmur most distinct at the apex, being transmitted to the axilla, and slightly to the aortic region.

June 1st. Nitro-glycerine stopped. Spartein sulphate and digitalis continued.

June 9th. Patient's general condition much improved.

June 14th. Improving. The voice is stronger. The ends of the fingers are clubbed. The second pulmonic sound is accentuated.

June 30th. Digitalis and spartein stopped, and strychnine sulphate (gr. $\frac{1}{100}$) three times daily ordered. Since the 21st instant the patient has been sitting up in a chair.

August 3d. On account of the child being too active, it was found necessary to keep her in bed. She has severe pains over the heart at night. Ung. belladonnæ was applied, and nitro-glycerine (m. $\frac{1}{100}$) was given every two hours when necessary.

August 11th. Patient has improved. Strychnine sulphate (gr. $\frac{1}{60}$) and fluid extract of digitalis (m. $\frac{1}{8}$) were given three times daily.

November 27th. The patient has been sitting up in bed for some time. She is in a fair condition, and appears to enjoy life. Is very pale, and becomes fatigued easily. The least exertion throws her into a perspiration. The heart and lungs have been in about the same condition for some weeks. Very little air enters the lungs.

November 29th. The child was taken suddenly, after dinner, with severe dyspnœa, rapid and irregular pulse, marked cyanosis of the face and hands, and expired in a few minutes.

POST MORTEM EXAMINATION.

Rigor mortis marked. Body emaciated. Very little adipose tissue present. The lungs were congested and compressed. The liver was somewhat larger than normal. The heart was enormously enlarged, weighing fifteen ounces. The heart shows universal chronic pericardial adhesion: Hypertrophy and dilatation of the left ventricle, and small vegetations on the aortic and mitral valves.

913 SIXTEENTH STREET.

A CASE OF CONGENITAL PHARYNGO-ŒSOPHAGEAL STENOSIS.

BY SAMUEL S. ADAMS, M.D.,

Professor of Pediatrics, University of Georgetown; Visiting Physician, Children's Hospital, Washington, D. C.

G. S. B., colored, female, aged four months, was admitted to the Children's Hospital, April 27, 1896. The mother stated that until two weeks ago the infant was in good condition, but at that time an inability to swallow liquids was noted, the milk being at once regurgitated through the nose. When admitted, the infant was in a comatose state, much emaciated; the pulse was rapid and feeble, and respirations were rapid and superficial. Hypodermatic injections of whiskey and strychnia were at once given, and external heat applied. In a few hours the child reacted. Attempts to administer food substantiated the statements of the mother, for regurgitation at once occurred. Attempts to pass even the smallest catheter proved futile. Then an attempt was made to locate the obstruction by introducing the finger well down into the œsophagus, but it had just passed the epiglottis when the point of resistance was encountered.

Nutritive enemata and stimulants were given at regular intervals, but the child emaciated rapidly. Life, however, was prolonged until May 11th, when the child died of exhaustion.

Necropsy, ten hours after death. Body very much emaciated, tissues pale and anæmic. Thoracic and abdominal viscera in an advanced state of decomposition. The stomach was empty, and appeared to be atrophied.

The upper part of the œsophagus, at its junction with the pharynx, ended in a blind pouch.

Dr. D. S. Lamb, of the Army Medical Museum, made the following report: "Tongue, larynx, portions of pharynx, œsophagus and trachea: Just about the pharyngo-œsophageal junction the pharynx ends in a wide cul-de-sac, except for a pinhole opening, which may have been made in removing the specimen or in opening up the œsophagus. The irregular upper border of

the opened œsophagus is of the same width as is the tube lower down, as if there had been a correspondingly wide cul-de-sac here. The specimen does not show any communication between the pharynx and œsophagus, direct or indirect. There is no evidence of inflammation from any cause."

COMMENTS.

Just how this child could have lived four months is most astonishing. That the correct age was given was subsequently verified by the birth return filed at the Health Department. It was my opinion that this was a case of acquired stricture from the ingestion of some irritant, but this has been negatived by the absence of evidences of preëxisting inflammation. An operation was deemed inadvisable owing to the extreme debility of the child.

The value of nutritive enemata in prolonging life is demonstrated in this case, for the child was sustained by them for fourteen days.

1 DUPONT CIRCLE.

TRANSPOSITION OF LARGE VESSELS IN THE HEART.

BY AUGUSTUS CAILLÉ, M.D.,

Professor of Pediatrics, New York Post-Graduate Medical School and Hospital; Visiting Physician to the German Hospital, New York.

The infant from which this specimen was taken was born a blue, or cyanotic child, and died when six weeks old. During its life it was moderately cyanosed; its pulse and respiration were rapid, and it coughed frequently. A heart murmur was not heard.

At the autopsy the heart was found in a normal position. The right ventricle, which was hard and contracted, showed a very thick, muscular wall. The left ventricle was found to be dilated, with moderately thick muscular walls. In place of the pulmonary artery there rose from the right ventricle the aorta, with its characteristic valves. It gave off the anonyma tubelaria and carotis, and pursued its course as arcus aortæ and aorta rescendent. The pulmonary artery took its origin from the site of the aorta in the left ventricle, and divided into two great branches which enter the lungs. The ductus botalli was obliterated, the ventricular septum closed, the foramen ovale in the auricular septum open. Tricuspid and mitral valves were normal, also the large veins.

Notwithstanding the transposition of the large arteries, a normal development of the fœtus in utero was possible as long as the ductus botalli remained open. After the closing of the ductus botalli, probably the only communication between pulmonary and general circulation possible was through the open foramen ovale. This foramen, as shown in the specimen, was also about to close, so that further life became impossible.

753 MADISON AVENUE.

ADHESIVE PERICARDITIS WITH COMPLETE OBLITERATION OF THE PERICARDIAL SAC, IN A CHILD SIXTEEN MONTHS OLD.

BY L. EMMETT HOLT, M.D.,

Professor of Pediatrics, New York Polyclinic; Attending Physician to the Babies' and to the Nursery and Child's Hospital, New York.

The specimen presented to the Society was removed from a female child sixteen months old, dying in the Nursery and Child's Hospital from acute broncho-pneumonia. During the last illness there was nothing to call special attention to the heart, and the condition found at autopsy was not suspected during life.

Besides the lesions of acute pneumonia, there was found an old pleurisy with extensive fibrous adhesions over both lungs.

The heart was somewhat hypertrophied, and weighed one and a half ounces. The pericardium was as thick as heavy blotting paper. The pericardial sac was completely obliterated. The adhesions between the two surfaces were tough and fibrous, and could not be torn apart without a good deal of force.

The family history threw no light upon the origin of the disease. The case was of interest chiefly on account of the age of the patient.

15 East Fifty-fourth Street.

DISCUSSION ON CARDIAC LESIONS.

Dr. W. P. Northrup referred to two cases of congenital cyanosis due to stenosis of the pulmonary orifice with an interventricular foramen. One of these died of cerebral abscess.

Dr. T. M. Rotch.—In presenting specimens we should speak as much as possible of the clinical symptoms. I have seen two cases where I was inclined to aspirate in the back for pericardial effusion, as the dullness was marked between the angle of the scapula and the spinal column. I did not, however, do it, as the effusion was rapidly absorbed.

Dr. Wm. Osler.—I may say that Ewart has quite recently described the sign to which Dr. Rotch refers in an admirable

paper in the *British Medical Journal*, March 21, 1896. He figures the spot of impaired resonance mentioned by Dr. Rotch in the infra-scapular region. Nothnagel, many years ago, called attention to the occurrence in pericardial effusion of a flat tympany in the axillary region, and of tubular breathing.

Dr. Osler then referred to the frequency with which acute pericarditis in children was overlooked, and to the recent work on the subject of adherent pericardium, which enables us to make the diagnosis more frequently than heretofore.

DR. AUGUSTUS CAILLÉ.—I would remark that there are several cases on record in which the purulent pericarditis with posterior dullness has been mistaken for empyema.

DR. A. H. WENTWORTH mentioned two cases of pericardial effusion which he had seen recently, in neither of which was there dullness behind the sternum, although the pericardial sac contained 90 c.c. in one case and 60 c.c. in the other at the autopsy. In both cases the border of the right lung extended to the median sternal line, and contained air. This probably masked the dullness.

[A pathological specimen showing perforate septum ventriculorum was presented by Dr. J. P. Crozer Griffith. For full description see ARCHIVES OF PEDIATRICS, September, 1896. A specimen showing a congenital heart lesion was also presented by Dr. Frederick A. Packard.]

A REPORT OF FOUR CASES OF ADHERENT PERICARDIUM IN CHILDREN.

BY GEORGE MONTAGUE SWIFT, M.D.,

Attending Physician to St. Mary's Free Hospital for Children, New York,

AND

ROWLAND GODFREY FREEMAN, M.D.,

Pathologist to St. Mary's Free Hospital for Children, New York.

Cases of adherent pericardium in children although probably not rare are apparently frequently overlooked. Our knowledge of this condition has received very valuable accessions recently from Broadbent in England.

Adherent pericardium arises from a single attack of pericarditis or from repeated attacks which may have a sub-acute character. The adhesions may be partial or complete. A marked hypertrophy and dilatation of the heart often accompanies this condition, although in some cases the heart remains normal in size or atrophied. Symptoms arising from embarrassment of the circulation due to this condition are dyspnœa, œdema, ascites, and vomiting.

The physical signs of adherent pericardium depend on the extent and position of the adhesions and on whether they involve only the two layers of the pericardium, or exist between the pericardium and chest wall or adjoining pleura, diaphragm or other parts of the mediastrinum. Of the physical signs often found the following are important:

1. Marked enlargement of the heart is present in many cases, accompanied by various murmurs.
2. Systolic depression at site of apex beat.
3. Systolic retraction of lateral and posterior walls of thorax.
4. Impeded descent of diaphragm in inspiration.
5. Dilatation of the veins of the neck with sudden emptying in diastole.
6. Absence or feebleness of apex beat.

In three of the following cases (i., ii , and iv.), the adhesions of the pericardium were complete and were associated with marked

cardiac hypertrophy and dilatation and gave double murmurs at both the apex and base of the heart. In none of the cases was a history of a previous attack of pericarditis obtained.

CASE I.—We find the following notes concerning Lizzie C., six years, admitted to St. Mary's Free Hospital for Children, New York, May 5, 1886:

The previous history was that the child for four years had been the victim of rheumatism, and that two years ago a physician had said she had heart disease. Upon admission she had a peculiar cachectic look; her abdomen, feet and legs were swollen and œdematous; there was great dyspnœa. The heart's action was turbulent. She was immediately tapped and three pints of turbid serum were drawn from the peritoneal cavity, and the cavity was then allowed to drain; it was then found that the lower edge of the liver was on a line with the umbilicus. Pericardial dullness extended from about an inch below the left clavicle to the abdomen; there was dullness laterally from two inches internal of the axillary line to the right of the sternum. There were double murmurs at both apex and base of heart, the point of maximum intensity being in the second left interspace; the apex beat could not be located. With inspiration there was depression of the sides of lower part of chest; there was no "pitting" of intercostal spaces over the pericardium. Posteriorly there was exaggerated resonance over upper portion of chest; there were râles over the lower portion of chest on both sides; there was some cough, but no especial pain.

The diagnosis was adherent pericardium, dilated heart. Because of the peculiar cachectic appearance of the child, the large liver, and the turbid, ascitic fluid, it was also thought that there was some malignant abdominal disease.

Death, May 14th. Autopsy same day; abdomen was distended with turbid fluid; liver large and dark with thickened capsule; lower edge almost on a line with umbilicus; upon section the liver was hard and firm with increase of interstitial tissue; the spleen was congested, but not especially enlarged; the cortex of the kidneys was thickened; the urinary examination during life had been negative. Upon opening the chest the pericardium was found firmly adherent to the chest wall and to the heart itself; the heart was much enlarged and dilated, concealing almost entirely the left lung; there was thickening of the endocardium, but no especial valvular lesion beyond some vegetations

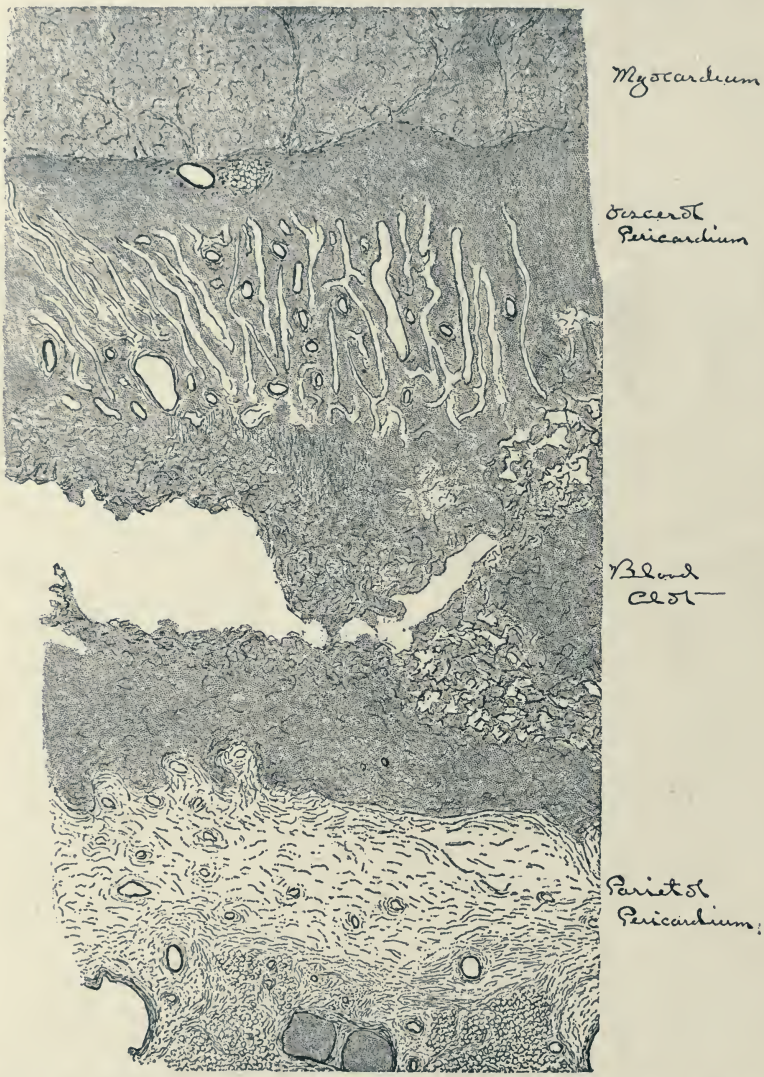
on the mitral and tricuspid valves; there was no pleuritic effusion; the lungs were œdematous.

The autopsy was not made by a skilled observer, so that the notes are meagre; but there can be no doubt, it seems to me, that the case was one of those described by Osler.

CASE II.—Emil, four years, admitted July 19, 1886. No history of rheumatism, none of scarlet fever. Has been sick about one month with cough, fever, pain in right side; looks sick, thin, emaciated; has been blistered on left side; on both sides, especially left, crackling râles; no other physical signs. Much dyspnœa; breathing so frequent and rude that heart-sounds are not heard. Two days after admission had acute pneumonia. About the ward on August 12th and seemed to be doing well; temperature has been irregular. August 18th, again sick with fever, frequent respiration, restlessness; cough, pain in side, location not definite. August 19th, some depression of left side; over heart irregular protrusion and depression of intercostal spaces; distinct recession of lower chest wall upon inspiration; boy pale and anxious looking. August 21st, condition better, but weak. September 1st, inspiratory recession of chest wall has disappeared, child seems better and has no elevation of temperature, but is restless at night and has dyspnœa. September 11th, much worse; face puffy, dyspnœa worse, restlessness increased, some pain; died September 13th. During his illness auscultation of heart was negative, while the apex beat could hardly be made out.

Autopsy September 13th. Chest alone examined. Pericardium adherent to both pleural, and distended two and a half inches across. Lungs adherent to chest wall; small vessels on pericardium distended, giving it a bright red appearance. Upon section about three ounces of purulent fluid escaped; pericardial sac about four times as thick as usual; within, the heart was covered with a thick mass of fibrin, and the heart was firmly fastened to the lower part of the pericardium which was in turn adherent to the diaphragm; the visceral layer of pericardium also much thickened. Valves normal; lungs normal.

CASE III.—Florence, fourteen years old, was admitted to St. Mary's Hospital, in June, 1895. No record of earlier condition or duration of sickness. She was at the summer home at Rockaway Beach during the summer, and was very comfortable on her return to New York. During the autumn she was attended



SECTION THROUGH MYOCARDIUM AND BOTH LAYERS OF PERICARDIUM IN CASE III., SHOWING HEMORRHAGE IN PERICARDIAL CAVITY, DILITATION OF THE BLOOD VESSELS OF THE VISCERAL PERICARDIUM AND ŒDEMA AND MYXOMATOUS DEGENERATION AT THE JUNCTION OF MYOCARDIUM AND VISCERAL PERICARDIUM.

at her home, but as her heart began to fail, she was readmitted to St. Mary's Hospital on February 8, 1896, and died February 20, 1896. Her symptoms were simply those of advanced cardiac disease together with double murmurs (to and fro) and friction sounds. There was fluid in the peritoneal cavity. The liver was somewhat enlarged.

Autopsy. February 21, 1896, twenty-four hours after death. Poorly nourished. Rigor mortis present. Twenty ounces of serum were found in the peritoneal cavity, and about fifteen ounces in each pleural cavity.

On opening the thorax a very large pericardium was about all that was visible. The parietal and visceral portions of the pericardium were everywhere adherent. The heart was dilated and hypertrophied. It was six inches in length and five and a half inches in its greatest transverse diameter. On section through the layers of the pericardium and the heart muscle, one sees between the parietal and visceral pericardium what appears to be inflammatory tissue, while between the visceral pericardium and heart muscle there appears to be some soft tissue which looks œdematous.

Microscopic Examination. (Fig. 1.) Shows hemorrhage into the pericardial cavity, marked enlargement of the blood vessels of the visceral pericardium, and œdema. Some mixomatous degeneration at the junction of the visceral pericardium and myocardium. No valvular disease found.

Lungs. Chronic congestion.

Microscopic Examination. The chronic congestion of heart disease with some interstitial change.

Liver. Much enlarged. Lower border at level of umbilicus, fatty.

Microscopic Examination. Congested and fatty.

Spleen. Negative.

Kidney. Cortex thick. Capsule not adherent.

Supra-renals and pancreas are negative.

CASE IV.—May Klein, aged ten years. The only previous history obtainable on admission was that she had had an attack of pleuro-pneumonia two or three months before. On admission was suffering much from dyspnœa.

Physical Examination. There was fluid in both pleural cavities. The cardiac area was much enlarged, and there was a to and fro (double) murmur at the situation of both apex and base.

No pitting of intercostal spaces. The abdomen was somewhat distended with fluid, but the lower edge of the liver, reaching almost to the line of the umbilicus, could be distinctly felt. There was some œdema of the lower extremities. Rest in bed and stimulation relieved the urgent symptoms much, but the child died at the end of a few weeks.

Autopsy April 14, 1896, twelve hours after death. Rigor mortis present. Body poorly nourished. Visceral and parietal pericardium everywhere adherent.

Heart. Much enlarged, being both dilated and hypertrophied, measuring five inches in length and four inches in its greatest transverse diameter. Heart valves show no vegetations.

Microscopic Examination. Marked interstitial myocarditis.

Lungs. Posterior portion of lower lobes congested.

Microscopic Examination. Chronic congestion of heart disease, with an acute pueumonic process added. Twenty ounces of brown fluid in peritoneal cavity.

Liver. Enlarged. Lower border one and one-half inches below from border of ribs. Mottled, yellow and red color with nutmeg appearances. Fatty and congested.

Microscopic Examination. Marked congestion and very fatty. But little liver tissue remaining.

Spleen. Not enlarged hard.

Microscopic Examination. Very marked congestion.

Kidneys pale.

Microscopic Examination. Chronic diffuse nephritis.

Pancreas. Very hard.

Microscopic Examination. Fat necrosis surrounding the pancreas.

Supra renals negative. Intestines not examined.

TEMPORARY INSANITY FOLLOWING TYPHOID FEVER.

BY SAMUEL S. ADAMS, M.D.,

Professor of Pediatrics, University of Georgetown; Visiting Physician, Children's Hospital.

Washington, D. C.

The four cases of temporary insanity following typhoid fever which I present, are unique and instructive. The rarity of this condition probably accounts for its omission from the standard text-books on the diseases of children. Sachs, however, observes that the infectious diseases are sometimes the exciting causes of insanity in children. These patients were all seen in the Children's Hospital.

MELANCHOLIA.

J. A. N., white male, aged seven years, was admitted to the hospital, August 2, 1895. The family history, as far as the boy's illness was concerned, was negative. He had recently recovered from typhoid fever. Before the fever he was as bright and quick as any boy of his age, but during convalescence it was noticed that he became uncommunicative, and lost interest in things about him. About a week before admission he ate crabs, snow-balls, and water-melon, the result being "cramps." On the third day after the feast he had convulsions, and lay in a stupor for forty-eight hours. The boy is poorly nourished and anæmic. He lies in apparent stupor, but has an intelligent expression when aroused. The pupils and reflexes are normal. Pulse and temperature are normal. Other systems normal.

August 7th. Lies in about the same condition. Numerous attempts to excite him proved failures. We talked to him pleasantly, scolded, pinched, attempted to startle him by quick movements and speech, all of which he treated with the utmost indifference. He was neither irritable nor peevish, but was happiest in his quietude.

His appetite was excellent, and he enjoyed food and drink, but never asked for either.

From his actions and our repeated failures to elicit any response, I rejected the statement that he was ever bright and

cheerful, and pronounced him to be "non compos mentis." His audition was all right, as he would cast his eye in the direction of sounds.

August 26th. He is walking about the ward, and takes his meals regularly and heartily, but still maintains his indifferent air and silence.

September 4th. Very little change in his condition. He obeys simple commands in a tardy, indifferent manner.

September 25th. Seems to be more interested in things about the ward, but no one has heard him utter a word. His friends were advised to place him in an institution for the feeble-minded, so they took him from the hospital.

Some weeks after his departure from the hospital, I learned that soon after his return home he began to talk intelligently and had fully recovered.

MELANCHOLIA.

William P., white, aged seven years, was admitted to the hospital, September 4, 1894. His father is well, but his mother died of pleurisy. His brothers and sisters are well. There is no history of nervous disorders in the immediate or collateral branches of his family. The boy was always robust, strong, and healthy until July 4th, last, when he was taken with typhoid fever, which lasted seven weeks. There were six more cases of the disease in the house in which this boy lived. It has been noticed that he has not gained strength since the fever subsided.

His general condition was bad, being poorly nourished, anæmic and weak. Physical examination showed negative results as to all systems except the nervous. The injury in the brain seemed to be confined to the intellectual faculties, as there was neither paralysis, hyperæsthesia, nor anæsthesia detected.

For the first few days he sat up in bed most of the day and night, rocking back and forth, picking his lips and scratching his head. Whenever anyone spoke to him he would repeat one sentence over and over again. His voice was whining and monotonous. His movements were all aimless. Every little while he would grow suddenly very quiet, but would soon begin screaming and shrieking, which it was impossible to stop, but ceased suddenly and without apparent cause. He begged for "something to eat" continuously, even though his hands were full of food at the time.

He was kept in bed about ten days without any perceptible

mental improvement. He did not seem able to concentrate his thoughts on anything but food. In fact, he did not seem capable of retaining any other idea in his mind much longer than it would take to tell him.

He soiled his bed during this period. When he was first dressed he had no idea how to get his clothing on, and had to be watched to keep him from undressing. When he was permitted to walk about the ward, the attacks of screaming became less frequent and less severe, but the same monotonous repetition of one remark kept up. In a very low and confidential tone he would say: "Give me a piece of bread and say," and repeat it in exactly the same tone twenty or thirty times. Whenever a doctor or nurse entered the ward he would trot along after him or her and plaintively repeat his request.

His pitiful and earnest begging for food induced me to direct that he should have as much bread and butter as he could eat. At my next visit the physician reported that he ate and ate, until it was thought he would injure himself, so the order was countermanded.

In a few weeks improvement was noticed. He would sit quietly in a chair for some time and watch the children play. Then he began to talk connectedly and intelligently, but soon grew tired if asked many questions, and became sullen. The nurses were instructed not to ask him many questions.

He was never inclined to resist any treatment, nor did he attempt to injure any one. In his darkest hour the suggestion of food would cause an expression of delight in his solemn countenance.

The mental improvement apparently began with relief from hunger, although he was never known to refuse food. He gained flesh rapidly and was soon entirely well.

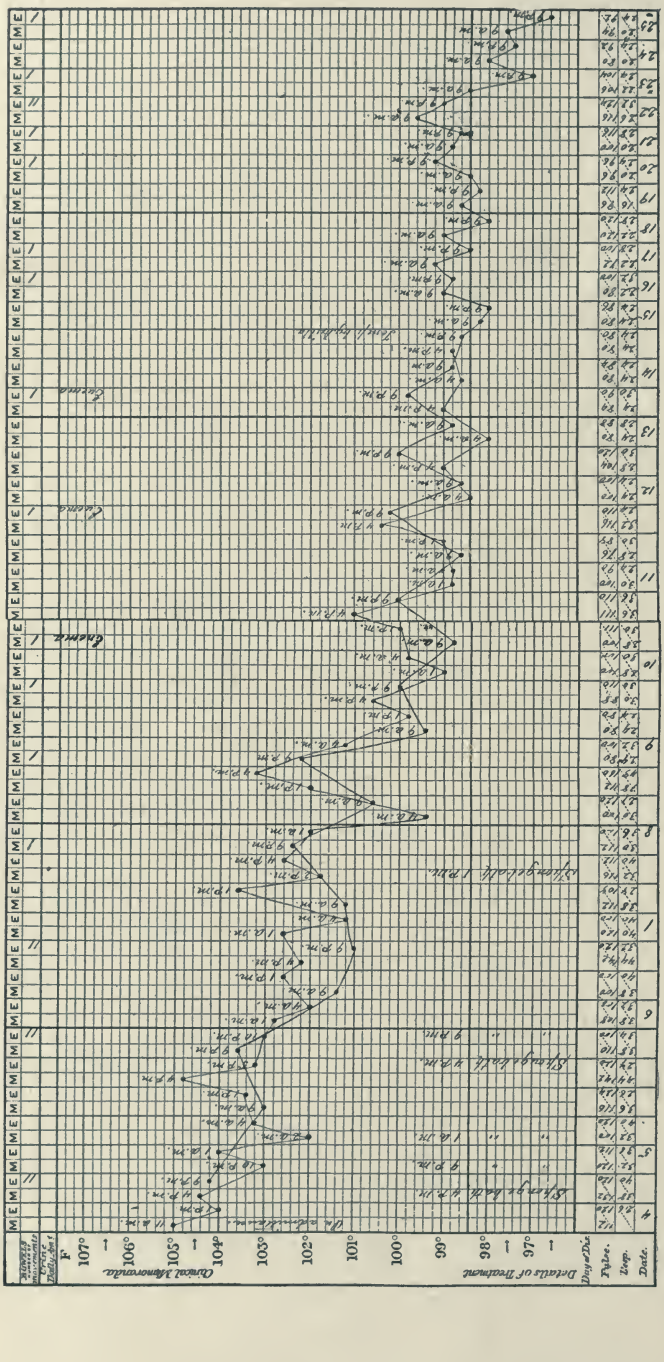
In his restoration to health, he was deemed to be unusually intelligent and attractive.

Some weeks after his discharge, he visited the hospital, but had no recollection of the fancies he had had during his treatment in the institution.

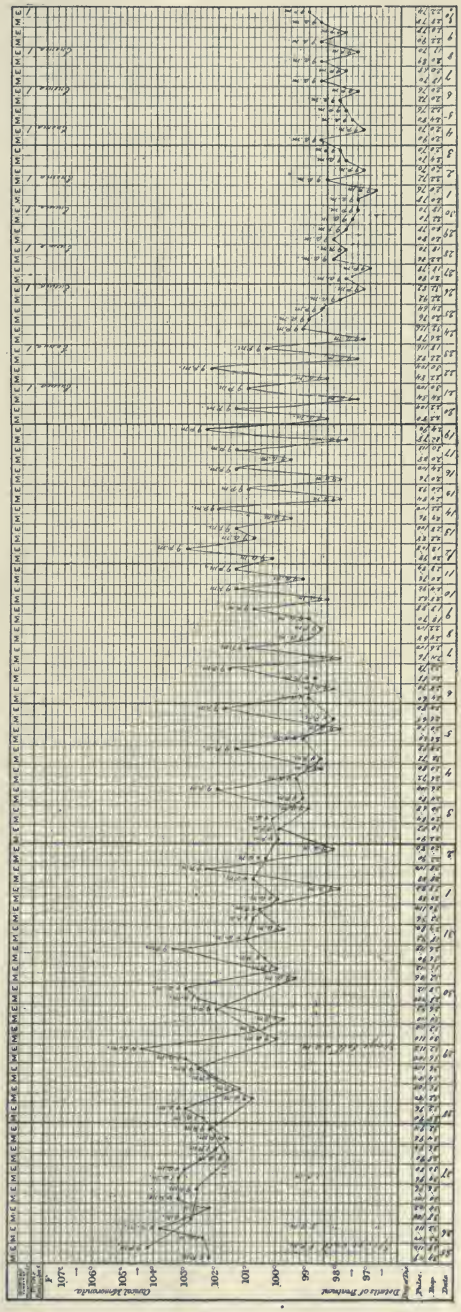
MANIA.

J. N., colored male, aged twelve years, was admitted to the hospital, September 4, 1895.

Father is in good health; mother is an inebriate; but the rest of the family history is negative.



TEMPERATURE RANGE.
Case III.—Typhoid Fever with Mania.



TEMPERATURE RANGE.
Case IV.—Typhoid Fever with Mania.

Has had pertussis and scarlatina. Has been in bed for one week. Has chills at times, vomits, and complains of pain in abdomen. Cannot sleep. Constipated. During the first few days after admission, was delirious at times, but took his nourishment quite well and slept during the night, occasionally requiring bromides and tincture hyoscyamus.

As shown by the chart, the patient was in the last week of typhoid fever, and the high temperature was controlled by an occasional sponge bath.

September 14th. The temperature reached normal and did not rise above that point thereafter.

September 18th. He has seemed to improve the past few days, and has slept well at night. He has taken and digested his food. At 10 P.M. he became restless, then alternately screamed and talked incessantly, A dose of sodium bromide (ten grains), and tincture of hyoscyamus (ten drops), soon quieted him, and he slept the rest of the night.

September 20th and 21st. He talks almost incessantly, has the most curious hallucinations, kneels at the foot of his bed, preaches and sings with great fervor, in the characteristic negro manner.

He called the day nurse to him in the morning, bade her sit on the edge of his bed, and related in a thrilling and confidential manner the adventures of the night nurse. He declared the doctors had come into the ward during the night and had beaten, cuffed and dragged the night nurse over the floor. Then he would call the children around his bed, and recite to them his adventures. The doctors, nurses and himself had gone out on the lawn and "dug, and dug, and dug all night and what do you think we found, a s-i-l-v-e-r-q-u-a-r-t-e-r." There was nothing profane in his conduct, and he was neat about his person and bed.

September 22d. He was ordered semi-solid food. His mental condition about the same.

September 24th. His mind clearer and the hallucinations less frequent. He sat up a while during the day. Solid food ordered.

September 26th. Mind seems quite clear. Occasional hallucinations, which quickly disappear when attention is called to some other subject.

Improvement continued and in a short time he was well and was discharged.

MANIA.

W. H., white, male, aged nine years, was admitted to the hospital, August 25, 1892. The family history was negative; his condition previous to the present illness had been fair; and there was no trace of nervous disorders in his brothers or sisters. He had been sick two weeks before admission with chilliness, fever and headache. His appetite had been capricious and his bowels constipated.

He was well nourished, but anæmic. Typhoid fever was diagnosticated, which was moderately severe and somewhat prolonged. The daily excursion of the temperature was sufficient to permit the slight delirium to be absent a greater part of the day. The delirium was always absent when the temperature was reduced by cool spongings or when it fell below 101° F. naturally.

Nothing unusual was noted until September 6th, when the boy became quite emotional, worried about his condition, tried to get out of bed, deceived the nurses, and talked at random.

His mental condition grew rapidly worse while his physical improved. He would take two quarts of milk and four ounces of beef-juice daily, but yelled for more.

September 27th. His temperature has not been above normal since the 24th, but his mental condition is much worse than when fever was present. He is now nervous, restless, and at times, maniacal. He will kneel at the foot of his bed and preach until he falls back from exhaustion. He alternately cries, prays, laughs, swears, and sings the same hymn over and over. He slanders the nurses and doctors in a confidential but artful manner. His memory is bad, ideas being retained but a moment. He sucks his teeth, tears his clothing and bedding into shreds, and urinates and defecates in bed. He pummels, bites, scratches, and spits on those approaching the bed. It is difficult to restrain him even in harness, for he twists, tears, and bites himself loose.

October 11th. There is no perceptible change unless that he is more emotional. He pays very little attention to what is going on about him. He does not distinguish persons, calling anyone "mother, sister," etc., and is especially affectionate to those he imagines to be friends and relatives long since dead.

Under the great mental strain he has become very much

weaker, but ingests and digests an enormous quantity of solid food in addition to the two quarts of milk.

October 17th. He has grown stronger, his general condition has improved, and he is a trifle more intelligent and reasonable. His paroxysms of emotion persist, his profanity is astounding and, at times, even the harness cannot restrain him.

It being impossible to properly manage him in the hospital his parents were advised to place him in an institution for the insane. Instead of following this advice, however, he was removed to his home where he ran amuck for the next few weeks, doing irreparable damage to person and property.

Within a couple of months from his discharge he fully recovered, and is now a promising youth.

1 DUPONT CIRCLE.

DISCUSSION.

DR. FLOYD M. CRANDALL.—I attended, four years ago, a young girl during a serious attack of typhoid fever. During the second and third weeks she was in a condition of stupor. As the fever subsided in the fourth week the stupor slowly disappeared, and she began to have delusions and hallucinations. She saw strange figures upon the wall, and animals about the room and on the bed. She refused to take medicine, and for a week it was with the greatest difficulty that she could be induced to eat. She required constant watching. This condition continued for about four weeks and disappeared gradually. Recovery was finally complete, and without the slightest mental impairment.

DR. WM. P. NORTHRUP.—I have never seen this condition in children. It must be rare under twelve years.

DR. JAMES C. WILSON.—I recall two cases of insanity in a maniacal form, following enteric fever. One patient was a girl of thirteen, and the other a girl of nine years. Recovery took place in the course of a few weeks in both cases. The condition seems to be that of nutritional insanity, and I myself have suggested the analogy to some forms of mania following starvation. I may mention incidentally that on Friday last I was in consultation with a physician in Philadelphia, in a thoroughly well characterized case of enteric fever, in a child seventeen months old, with well marked rose spots, enlarged spleen, and typical infantile remittent temperature. The case was at the twelfth day of the attack.

DR. WILLIAM OSLER.—Except in hemiplegia, a favorable prognosis can be given in nearly all the cerebral affections fol-

lowing typhoid fever in children. Slight melancholia is not very uncommon, and a form of aphasia; sometimes there is a sort of stupidity left for months after the illness, a condition similar to that which Dr. Adams has described in one of his cases. The outlook for the post-typhoid insanity is good. It is most exceptional for the patients to remain permanently insane.

DR. W. S. CHRISTOPHER.—Aphasia has been mentioned as a not very uncommon complication of typhoid fever. I have seen aphasia occur during the third week, when the fever was at its height. This patient was two years and one week old when the disease began. It was as severe and typical a case as I have ever seen recover. The temperature on the first day was 104.5° and 106.5° on the third day. The aphasia was complete, and lasted about three weeks. Recovery was complete.

DR. SAMUEL S. ADAMS.—I agree with the President, that this condition is due to nutritional disturbance, and infection has nothing to do with it, except in causing primarily, the nutritional disturbance. As Dr. Osler says, "the prognosis is favorable, and as soon as the patient is properly fed it recovers." I contend that we put off giving solid food too long. It should be given a little earlier than is the custom.

THIGH FRICTION IN INFANTS UNDER ONE YEAR OF AGE.

BY CHARLES W. TOWNSEND, M.D.,

Physician to Out-Patients, Massachusetts General Children's, and Boston Lying in Hospitals, Boston.

Although Jacobi called the attention of the profession to masturbation in infants some twenty years ago, the fact that its occurrence in infants is not mentioned in most text-books, and that in the following cases the true nature of the trouble was not understood by the parents and by physicians previously consulted, has led me to report these cases. The five cases all began in infants under one year of age, at which time it seems incredible that the sexual appetite should exist.

CASE I.—Etta D., twelve months old, seen at the Children's Hospital. At the age of eight months the infant was noticed crossing her right thigh over the left, closing her eyes and clenching her hands. After a minute or two this action was followed by complete relaxation, with sweating. These "attacks" as the mother called them, occurred about once a week until four days ago, since when they have occurred as often as every half hour during the day.

The infant was kept in the waiting-room until an attack occurred; the right thigh was forcibly flexed over the left, thus pressing on the vulva, and was held there rigidly, with, at the same time, slight rotary movements. The infant became flushed. The act ceased abruptly after a minute or two, and the infant lay back relaxed and perspiring. The nature of the act was unmistakable. The infant was well nourished and well developed and showed no irritation about the genitals.

CASE II.—Ida H., seen at the Children's Hospital. Is now three years old, but since the age of eight months, has at times pressed her thighs together, moving them up and down. The face is flushed at the time, but becomes pale and perspiring afterward. The "attacks" averaged two or three a week, although, sometimes, several occurred in one day. They are apt to come on when the child is tired and fretful, and appear to have a sedative effect. The mother was hysterical. The child was fairly developed and decidedly precocious mentally. There was no abnormal development or redness of the genitals.

CASE III.—Marian C., a cousin of the preceding case, the mothers being sisters. The history is kindly given me by Dr. W. N. Bullard, under whose care she came in the nervous department of the Children's Hospital.

Family History.—Father very nervous, mother subject to severe hemicrania and hysteria. Maternal grandfather subject to sick headaches and epilepsy. Two aunts, hysterical. No insanity. Child is now four years old. When six months old thrown from a carriage, struck head and was unconscious for two or three days. A few months later, before she began to walk, it was noticed that she would press with her hands on the lower abdomen and bend her body forward. This habit increased after an attack of whooping cough which debilitated her. Within the last three months she has masturbated with the hands and various objects.

CASE IV.—Molly S. The report of this case is taken from the Children's Hospital records, the case not being seen by me. The patient, now three years old, began, before she was a year old, the habit of crossing her legs at intervals and holding them tightly together for a few minutes. Flushing and sweating accompany the act. The child was described as the picture of health.

CASE V.—Ella C., seen at the Children's Hospital. Father well, mother of nervous disposition and has had rheumatic fever. Child now six years old. At the age of six months it was noticed that whenever she was placed in a high chair, she moved to the front of the chair and turned side wise, so that the edge of the chair came between her thighs. She would then move up and down, become red, and even purple in the face, sweat profusely and seem exhausted afterward. This was done several times daily, and, although the mother did not understand the significance of the act, she put away the chair. A little later, when the child was able to walk, she would act in the same manner with a small rocking chair. Not until the child was five years old did the mother finally awaken to the meaning of these actions, and by careful watchings and scoldings, she broke off the habit six months before I first saw her.

The child was brought for an attack of chorea, being the second attack, the first one occurring two years before. The child was very bright and well developed, but extremely nervous.

To sum up: All of these five cases of thigh friction began

during the first year of life, the youngest being six months old. All were in females, and in all, the nature of the trouble, although perhaps suspected, was not fully recognized by the parents.

76 MARLBOROUGH STREET.

DISCUSSION.

DR. S. S. ADAMS.—I am somewhat disappointed that Dr. Townsend did not give a plan of treatment. I have two cases under observation, and am perplexed to know what to do with them. The younger is eighteen months old, and has been treated since her birth by homeopathic physicians. At the sixth month she developed peculiar movements, of which the parents had never learned the significance.

She had been habitually constipated, requiring daily enemata or other mechanical assistance, since her birth. She was nursed to about the eighth month, and was subsequently fed upon milk. When I first saw the child she presented a picture that I had never seen. She was in her mother's arms and seemed to be greatly excited. She was now placed upon the floor. She immediately ran across the floor to a chair, which she pressed on. She now fixed her muscles firmly, threw the right thigh across the left and, by bending the knee, went through an *up and down* motion. Her face became very red, but soon relaxation occurred when she would exclaim, with a sigh, *there!* Such *attacks* happened every five minutes.

I now made a careful physical examination, but failed to discover any irritation, especially about the genitalia. A complete urinalysis was also made. Attention was directed to the diet with the hope of overcoming the obstinate constipation without the use of local means.

Under strict *regimé* she improved during the first week, but was taken sick with malarial fever and passed under the care of her former physician.

The second case, a girl, aged two and one-half years, was brought to me from Maryland, with a diagnosis of mental deficiency. She was carefully observed and examined, and I concluded that the movements were choreic. My colleagues, Drs. J. Ford Thompson and Acker, and Dr. E. L. Tompkins, a neurologist, also examined the patient and concurred in the diagnosis.

She steadily improved under increasing doses of Fowler's solution, and in three months was discharged, practically well. In a short time, however, she was reported to have gone back to her former condition. Now her mother described the attacks so well that the diagnosis was changed to thigh friction. If she comes into the hospital again, I shall try some mechanical appli-

ance that will keep the legs separated. We should endeavor to stop the movements at the outset.

DR. LEROY M. YALE.—There is an apparatus which may prove of some use, but not if the child uses the hands. Dr. Adams will perhaps recollect the old shackle boards formerly used in the treatment for club foot. It consists of a foot board, with a long board between the heels and toes. The child can shuffle along, but cannot get the limbs together.

DR. A. H. WENTWORTH.—I have recently seen a girl of seven or eight, who obtained the same results without using the thigh friction, but by movement of the pelvis. Fixing the thighs would not prevent it in such a case.

DR. L. EMMETT HOLT.—I think a study of the urine of these cases is very essential to successful treatment. The irritation caused by a highly acid urine is often the beginning of masturbation. I have had cases that were relieved by simply attending to that alone. In young infants, I believe, there is nothing so efficient as mechanical restraint.

DR. WM. D. BOOKER.—Examination of the urine, as Dr. Holt has suggested, is important. Mechanical appliances irritate a child, and do not usually accomplish as much as careful watching and correction with mild punishments.

DR. GRIFFITH.—In my experience, cases of thigh friction in infants are of very unusual occurrence. A large number of infants are brought to the out-patient department of the Children's Hospital in Philadelphia, but neither there nor in the wards have I ever seen an instance of thigh friction until a year ago. This infant, a girl, exhibited exactly the symptoms which Dr. Townsend has described. They were so suggestive that I found that even the uneducated mother had had her suspicions aroused.

DR. TOWNSEND.—I have found in young babies and in beginning cases, the treatment by watching and corporal punishment referred to by Dr. Booker, very efficacious. In cases of masturbation in older children, a subject I have not included in my paper, mechanical restraint, watching and punishment are alike often ineffectual.

My object in presenting the paper was, not to consider treatment, but to call attention to the subject of masturbation in *infants*, as my experience has led me to believe that cases of this sort are misunderstood and overlooked by physicians and the laity alike. These cases, if recognized early, before the habit is formed, can, as a rule, be easily stopped, whereas, if they are left to go on until the child is four or five years old, the habit has not only affected the health of the child, but has become so firmly seated that it is almost impossible to dislodge.

A CASE OF SUPERFICIAL GANGRENE.

BY B. K. RACHFORD, M.D.,

Clinician to Children's Clinic, Medical College of Ohio, Cincinnati.

I ask the privilege of presenting to this Society the detailed history of a very remarkable case of superficial gangrene which occurred in my private practice last winter.

CASE.—A male infant; seven months old; has no teeth. He is the first and only child.

Family History.—The mother is a splendid specimen of womanhood, with a most excellent family history. She was never in the family way but once, and then gave birth to a normal child (my patient) at full term. The family history on the father's side is also good, but the father himself is not very well. He has had a tendency to chronic diarrhœa for years. Some five or six years ago (several years before his marriage), he had a sore on his lip, which was diagnosed to be a chancre. This sore readily healed under treatment, but he continued to take protoiodide of mercury, under the advice of his physician, for about three months. The father does not remember that he had any symptoms of syphilis following the initial lesion, but from my knowledge of the man, and the subsequent history of both himself and his child, I have no doubt but that the above diagnosis was the correct one.

My little patient is a very strong and well nourished boy, seven months old. I never saw him till his present illness, but his mother tells me that from his birth to the present time he has been perfectly well. "He has never cried an hour in all his life," and his appearance at the present time, as shown in photograph No. 1 (taken on the third day of his present illness), sustains the

mother in her statement that he has always been in vigorous health.

January 19, 1896. The mother and child, both apparently in perfect health, went for the night to the child's paternal grandparents.

January 20th. At 8.00 A.M. the baby was bathed and dressed and at that time was quite well and playful. About 9.30 A.M. he commenced to cry, and continued almost uninterruptedly for two hours in spite of all the walking and petting which he had during this time. At 11.30 A.M., the mother undressed the child with the hope of finding the cause of his crying. In this search she was shocked by finding a large "blue-black" spot, about the size of the palm of her hand, on his left buttock. This spot was almost circular, and as I afterward found by measurement, was three and a half inches in diameter in one direction, and three inches in the other. Soon after the spot was found and uncovered the child seemed free from pain. At any rate he ceased to cry; this may have been partly due to the fact that the spot was carefully protected after it was found; but there can be no doubt but that the child suffered more pain during the first two hours of his sickness than he did at any time subsequently in a corresponding period, during his long illness. On the discovery of this ominous looking spot, the mother was so frightened that she called in the nearest physician. I did not see the baby till 2.30 P.M., just five hours after he began to cry, and three hours after the discovery of the spot. During the first examination which was made in my office, the baby did not cry and was not at all fretful. I found him perfectly normal in every way except

FIG. I. PHOTOGRAPH OF PATIENT TAKEN ON THE THIRD DAY OF ILLNESS.

that there was a circular patch of superficial gangrene on the left buttock, three by three and one half inches in diameter.

The spot was exactly the same size as when discovered by the mother three hours before, and I may here add that it never increased in size afterward. When I saw the spot for the first time it was black, and there was no mistaking the fact that the skin was gangrenous. There was not the least evidence, either in the history or in the appearance of the spot, that would permit the suspicion that the gangrenous patch might be due to a burn or a trauma of any kind. The gangrene was perfectly dry, there were no blebs or blisters, and the line of demarcation was as distinct when I first saw the patient as it ever became afterward, and I may here add that throughout the history of this case the gangrenous patch remained so dry that it was a very simple matter to keep the sore surgically clean.

To the parents I made a diagnosis of superficial gangrene, and told them not to be surprised if a similar spot appeared on the opposite buttock, but the second spot never appeared, and the subsequent history of the case did not in any way resemble symmetrical gangrene. I prescribed a simple boracic ointment and sent the child home.

January 23d. It is now three days since the sudden appearance of the patch of gangrene, and my little patient has apparently, in every other way, remained in perfect health. He nurses, sleeps and plays as before, and never manifests any uneasiness, except when placed in a position that causes pressure upon or irritation to his left buttock. He will lie upon his back, happy and contented, playing with his toys, for hours at a time. Across his mother's lap, face downward, is another of his favorite positions. In these positions the sore spot is protected, and the baby is apparently unconscious of its existence. The gangrenous patch of skin looks very much the same to-day as it did when the child was in my office three days ago. The spot is perfectly dry, uniformly black, and exactly the same size as when it first appeared. The dead skin is perfectly smooth and shows not the slightest evidence of scratch or contusion. Along the line of demarcation the healthy skin is slightly inflamed and the dead skin shows a beginning separation. The sore is surgically clean, and can be dressed without protest from the baby.

I took advantage to-day of the only bit of sunshine which we

have had in three days, to get the photographs* here presented. They were taken with the child in his favorite positions above described. These photographs show the size, shape and location of the gangrenous spot better than it can be described. The almost circular shape of the spot is noteworthy.

January 27th. One week has passed since the trouble began, and the baby has remained well and apparently unconscious of the existence of the sore, except when it is dressed; at these times he is somewhat fretful, and may cry for half an hour afterward. The gangrenous skin has been slowly separating since

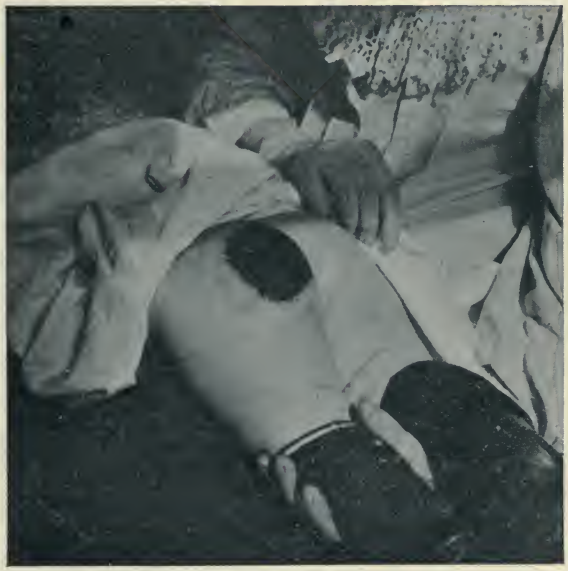


FIG. II.—SUPERFICIAL GANGRENE TAKEN ON THIRD DAY OF ILLNESS.

the third of January, so that at present it is separated from the normal skin by a circle of healthy granulating surface about one half inch in width. The sore is surgically clean and the gangrene perfectly dry. The temperature of the baby has been taken every day from the beginning, and has never been above normal. The father of the child is in bed to-day with the influenza. He has been sick for several days.

January 30th. Baby has influenza, fever 102.5° F., irritable

* These photographs were taken by Mr. Joel Ross, a student at the Medical College of Ohio.

stomach, cough and fretfulness. Sore is healthy; the gangrenous skin is slowly separating.

February 5th. Is convalescent from the influenza, has no fever, no cough, sleeps fairly well, but he has not recovered his amiable disposition. The gangrenous skin, which is now very loose and seems on the point of coming away, is acting as an irritant to the underlying and surrounding parts. The sore, for this reason, is now considerably inflamed and quite tender to the touch. The child cries bitterly when the dressing is changed.

February 8th. The dead skin came away on the afternoon

FIG. III.—SUPERFICIAL GANGRENE TAKEN ON THIRD DAY OF ILLNESS.

of the sixth of February (seventeen days from the appearance of the gangrenous patch), and since then the sore has not been so painful. During the past eight days the child has lost in strength and weight, and his general condition is nothing like as good as it was before he had the influenza. An examination of the dead skin showed the gangrene to have been very superficial, involving only the skin and a little subcutaneous tissue.

February 16th. The father of the child died to-day, after a

four days illness, from acute peritonitis. He was brought home, almost moribund, from Columbus, Ohio, where he had gone on business, a few days after recovering from the attack of influenza above-mentioned.

February 20th. The baby has made very little, if any, improvement within the last three weeks. He is pale, fretful, has little appetite and the sore is healing very slowly. He has no fever.

February 21st. He has a well-marked erythema intertrigo in the left groin, which the mother discovered when she dressed him this morning. He also has an erythematous rash over the back, neck and shoulders. I ordered calomel, to be followed by chalk mercury, one grain morning and evening. For the intertrigo I advised the local application of a powder of oxide of zinc and starch. For dressing the sore I continued the use of a simple boracic acid ointment, which had been used from the beginning.

February 28th. The intertrigo has disappeared, but the rash is still present. The sore is healing slowly and is now about the size of a silver dollar. Chalk mercury continued.

March 28th. The sore has healed, and, at the present time, a large red cicatrix marks its former site. The baby is now apparently in perfect health. The chalk mercury has been given continuously for five weeks, and there can be no doubt but that he has been materially benefited by the mercurial treatment. At this date the baby passed out from under my medical observation, and the mercurial treatment was discontinued.

On May 9th, just six weeks after the discontinuance of the mercury, the mother returned to my office with her child, because the erythematous rash had again appeared over his neck and shoulders, and had refused to yield to local treatment with an oxide of zinc and starch powder which the mother had been using during the past week. She says that he has been fretful for the past few days, but apart from that he has been well since she had him at my office, six weeks ago. At this examination the baby appeared normal in every way except the well marked rash on his neck and shoulders and the scar on his left buttock, which had changed very little. I again ordered calomel, to be followed by one grain of chalk mercury, morning and evening.

May 22d. The rash has disappeared, and, so far as the

mother's observations are to be relied upon, "the child is well." The chalk mercury was continued.

This case has been one of intense interest to me, and one the etiology and pathology of which I have not been able to understand. But there are certain facts and symptoms in this case which must form the basis for its etiological study, and these are as follows:

1. The father of the child had syphilis, the mother was perfectly well.

2. The child was apparently in perfect health up to the time (seven months), the gangrene occurred.

3. The gangrenous spot appeared suddenly, accompanied by pain but no fever.

4. The gangrene was dry and very superficial. It was confined to the skin and subcutaneous tissue, and was no deeper in the centre of the patch than it was at the circumference.

5. This patch was three inches in diameter and almost circular. It was the only patch that formed.

6. Every portion of the skin of the entire patch seemed to die at the same time, and the gangrenous spot did not increase in size after it was first observed.

7. Trauma and external pressure can absolutely be excluded from consideration in the study of the etiology of this case.

8. Sepsis, as a direct producing cause of the gangrene, can also be excluded, since the gangrene was dry, and there was never the slightest evidence that a local bacterial infection played any part in producing the gangrene.

9. The plugging of a small blood vessel could not have produced the gangrene, because the blood vessels of the skin are not terminal.

10. The sudden appearance of the gangrenous spot was probably the initial symptom of hereditary syphilis, which had been transmitted to the child by the father alone. The mother being and remaining absolutely free from this disease.

In the light of the above propositions, the only explanation which presents itself to my mind for the sudden appearance of the gangrenous spot in the manner above described, is, that a sudden vaso-motor spasm completely obliterated the blood vessels of this spot. This spasm was of course tonic and continued long enough to cause absolute death of the skin.

This explanation is unsatisfactory because we cannot under-

stand how so complete and so circumscribed a vaso-motor spasm could occur without apparent cause other than hereditary syphilis, but it must stand till a better or more satisfactory explanation is offered.

These cases of superficial gangrene may also be of value in the study of the circulation of blood in the skin.

323 BROADWAY.

CEREBRO-SPINAL MENINGITIS, FOLLOWED BY HYDROCEPHALUS—AUTOPSY.

BY W. P. NORTHRUP, M.D.

Professor of Pediatrics, Bellevue Hospital Medical College; Attending Physician to the Presbyterian, Foundling and Willard Parker Hospitals, New York.

A three-year-old boy entered Presbyterian Hospital, New York, with symptoms of cerebro-spinal meningitis. He had already been sick a month. Before this illness he had usually been well and strong.

His personal history was as follows: A month before entrance he was "out of sorts and feverish," the mother said; the next morning he vomited; the same evening he had six epileptiform convulsions; for the next few days neck and back were stiff, and he cried when moved. After a week his symptoms subsided, and for another week the boy seemed improved.

At the beginning of the third week of illness, the stiffness and pain in the neck returned. He had general spasmodic movements of the arms and legs, the light seemed to bother him, so the mother said; he ground his teeth, rolled his head; was stupid, called out, eyes had an unnatural stare; he lay in a stiff sort of way, did not speak, and cried when disturbed.

On examination at the hospital the following notes pertinent to the case were taken: Poorly nourished, and anæmic; dentition good; chest showing slight degree of rickets; practically no fever; tongue moist. He was very irritable; neck rigid, eyes staring, moderate photophobia; knees and thighs flexed while lying in bed; sensitive to pressure along vertebral spines; resistance and pain on motion of legs; free general desquamation. On abdomen and thighs a mottled, macular eruption, a little dusky in appearance. Abdomen retracted; heart rapid (120); lungs negative; pharynx and tonsils a little ingested; respiration 19, temperature 98° F.

For the first week in the hospital his condition remained much the same. Temperature irregular, the highest being 102.2° F. Spasmodic movements of left hand were mentioned in the history, and occasional vomiting. During the week he

gradually developed an abscess of the left side of the neck, with fever. After the abscess was opened, he continued the gradual improvement. Eight weeks after entrance to hospital, and the twelfth week of his illness, when apparently doing very well, he suddenly had a little attack which is difficult to explain. To quote the notes: "At 7 P. M. pulse became weak and intermittent; pupils pinhole; eyes did not move when lids were lifted. This attack passed off in five minutes, head and neck still rigid."

Following this his convalescence became established; he gained flesh, went about the wards, visited the roof promenade, and after six weeks was discharged cured.

The patient was then sick altogether four months. The diagnosis by the attending physician was epidemic cerebro-spinal meningitis—abscess of the neck—recovery.

One month from the date of his discharge from hospital, and five months from the first appearance of this disease, our young patient, now three and a half years old, re-entered the hospital with what appeared to be a return of the previous symptoms.

During his sojourn at home he had apparently regained his former health, flesh, and strength; but he never became so mentally bright as before his illness.

His history on second entrance was: Three days of vomiting of all food; frequent putting his hands to his head, and crying out as though in pain; no convulsions; pain frontal and temporal; vomiting projectile; no fever.

On readmission this time to my service his condition was much better than on first admission. Well nourished, not anæmic; tongue coated slightly; some erythematous spots on skin; head apparently larger; eyes were staring, restless; head held rigidly; mental dullness; few subcrepitant râles; heart normal.

His last stay in hospital was one month. In this time he screamed out occasionally, vomited at irregular intervals. He lay in stupor, quiet, with a pale, mask-like countenance. When his head was brought forward on his chest, his countenance showed signs of pain, but it was also observed that if his arm was flexed or extended, or his leg was flexed, in fact, if any of his muscles which had remained for hours unmoved were put upon a stretch, his countenance evinced pain. Suddenly, without any premonition, the child died.

Autopsy by Dr. Thacher.—Brain, convolutions flattened;

ventricles extremely distended four or five times their normal size, contents of ventricles clear serum; ependyma granular, no tuberculosis. Spinal cord, no tuberculosis; central canal not distended.

The post-mortem examination was made with tuberculosis in mind. No tubercles were found in the pia mater; the granular condition of the ependyma was proved non-tuberculous under the microscope; no tubercles were found in the bronchial lymph nodes; no cervical or mesenteric lymph nodes were tuberculous, and there was no bone tuberculosis.

To recapitulate: A three-year-old child with negative family and personal history ran a course of epidemic cerebro-spinal meningitis, from which he seemed, after four months, to have recovered. After one month of apparent bodily health his symptoms returned.

If there had been, as so frequently happens, no previous obtainable history, I should have pronounced this second attack somewhat confidently tuberculous meningitis with dilated ventricles.

The autopsy showed non-tuberculous ependymitis, the ventricles dilated and serum-filled. Here are explained the phenomena of the second illness: Intraventricular pressure, hydrocephalus, dilated and distended ventricles, giving rise to symptoms simulating the late stage of tuberculous meningitis.

As to the former illness, with its evidences of pain, its fever, stiffness of neck, and eruption, that already given would seem a reasonable diagnosis. At autopsy there was no trace of a former meningitis, no thickening of the pia, no adhesions, blood vessels apparently normal. There was a form of inflammation then, not tuberculous, but having products of inflammation which were absorbable. Our conclusion is that the patient first suffered from cerebro-spinal meningitis, a certain proportion of the lesions of which subsided. The lesion of the ependyma continued, the product of its inflammation being mostly serum. In other words, hydrocephalus was a sequel of cerebro-spinal meningitis. This is in accord with the observations and teachings of Dr. Delafield, and in accord with most classic literature.

This case seemed of interest on account of the rarity of such cases leaving a complete history.

57 EAST SEVENTY-NINTH STREET.

DISCUSSION.

DR. CHARLES W. TOWNSEND.—I reported a case some eight years ago of cerebro-spinal meningitis that lived three months, and finally died. On two occasions during that time the patient, a boy four years old, was apparently convalescent. Thus, from the twenty-fourth to the forty-seventh day of the disease, the temperature remained normal, and during the latter week of this remission the patient took food readily, talked and laughed. Again, from the seventy-first to the seventy-third day, with normal temperature, relaxation of spasms and other symptoms, the outlook seemed hopeful, but the disease returned, and the patient died on the eighty-seventh day. Such a long intermission of perfect health as in the case reported by Dr. Northrup is certainly of great interest.

Henoch states that the prognosis is very favorable in the intermittent cases observed by him in Berlin. He has never seen a death during an exacerbation or relapse.

DR. WILLIAM OSLER.—It is interesting that these cases in children are the exact counterpart of the chronic form of cerebro-spinal meningitis in adults, well described by Stille, Heubner, and others. In Vol. III. of *The Johns Hopkins Hospital Bulletin* I reported an interesting case of a child two and a half years old, ill for several months with cerebro-spinal meningitis, who made a good recovery.

DR. T. M. ROTCH.—I had a similar case where the child recovered, but the pressure caused blindness.

DR. OSLER.—Severe cerebro-spinal meningitis may recover completely. I know of a physician in good practice and clear-headed who had it, and is now quite well.

DR. A. H. WENTWORTH.—I think they recover clinically, but I doubt whether an inflammatory exudation can be so completely absorbed as to leave no trace microscopically.

DR. AUGUSTUS CAILLÉ.—In such cases I would draw attention to spinal puncture. I think it would be of value in any case to examine the fluid.

DR. W. S. CHRISTOPHER.—One phase of the subject which has interested me is the occasional occurrence of lymph on the meninges without the appearance during life of any evidence of meningitis. I have seen an extensive exudation over the whole cortex with absolutely no signs, during life, of meningeal disturbance.

DR. L. EMMETT HOLT.—I have seen that condition frequently. It is not very rare in children.

DR. CAILLÉ.—I have seen the brain covered with a greenish-yellow exudation that was never suspected during life.

DR. OSLER.—I have referred on several occasions to Stokes' dictum, which we all should lay to heart—that there are no specific symptoms of cortical meningitis. Take a case of so-called cerebral pneumonia; who can say during life whether or not there is exudate on the meninges? Clinically there is not a single differential criterion between the cases with perfectly normal meninges and those with a thick plastered exudate on the membranes. When the base is involved, or when with it the cervical meninges are attacked, one can often make a diagnosis of meningitis.

DR. NORTHRUP.—The case was interesting because it was a well-marked case of cerebro-spinal meningitis which made a recovery apparently. I have never seen any other case that pursued a course similar to this one. Tubercular meningitis was suspected.

AN UNUSUAL FORM OF CONGENITAL CARDIAC MALFORMATION.

BY T. M. ROTCH, M.D.,

Professor of Pediatrics, Harvard University.

This case is of considerable interest from a clinical as well as a pathological standpoint. It was a male, the first child of healthy parents. The labor was an easy one, and the infant was externally well formed and developed. It showed no cyanosis or abnormal symptoms of any kind, was vigorous, cried strongly, and nursed well.

As the parents were very apprehensive as to whether the child was perfectly normal, an unusually careful physical examination was made at the time of birth by Dr. Richardson, who delivered the mother, and twenty-four hours later by me. The skin was normal in color. Nothing abnormal was detected in the lungs or heart, the latter especially showing a normal area of dullness, clear, strong, valvular sounds and normal rhythm. The pulse, respiration, and temperature were normal.

Until it was sixteen days old the infant throve, and was seemingly perfectly healthy; it then refused to take the breast, and for a few hours in the afternoon seemed somewhat cold, was slightly cyanotic and had a temperature of 35.2° C. (95.5° F.), with quickened respirations. An examination of the heart and lungs at this time detected nothing abnormal. External warmth and stimulation soon restored the equilibrium of the circulation, and the infant soon nursed and seemed as well as usual.

Early on the following morning, however, the pulse and respirations became rapid; the temperature rose to 37.7° C. (100° F.); it refused to nurse, failed rapidly, and without any other symptoms or any especial cyanosis, died in the afternoon. Nothing abnormal, excepting that for a short and transient period a small area of dullness just below the left clavicle was detected.

The point of clinical interest in the case is that we should be very careful in our statements as to the physical condition of seemingly normal infants in the early weeks of life.

A post-mortem examination made within a few hours after death showed nothing abnormal, with the exception of a malformation of the heart and blood vessels. Nothing was found to account for the dullness under the clavicle.

There was a general streptococcus invasion for which no cause could be found. The cord had come away at the usual time without leaving any abnormal condition in the neighborhood of the umbilicus. An examination of the heart showed it to be decidedly hypertrophied, and the foramen ovale to be open. In the upper part of the interventricular septum there was an orifice about 1½ c. m. in diameter connecting the two ventricles. From the cavity of the left ventricle, what was apparently the pulmonary artery was given off, and from the cavity of the right ventricle in like manner the ascending aorta was given off.

This transposition of the great vessels is of course a common malformation in connection with defects in the interventricular septum. In following the course of the great vessels, an unusual distribution of their branches was found. What was supposed to be the pulmonary artery was found to dilate into a pouch.

From this pouch three vessels were given off. The two lateral ones went to the right and left lungs respectively. A central vessel, practically a continuation of the pulmonary artery, but seemingly the ductus arteriorus, passed upward and to the left and downwards, forming and taking the place of the arch and descending portions of the aorta. The first branches given off from this vessel were the intercostals, and the blood supply for the trunk and the lower extremities was entirely from this vessel.

What was supposed to be the ascending portion of the aorta was found to extend up on to the trachea and to then bifurcate into two branches—apparently the right and left carotids. These branches again gave off the subclavians; practically, however, the left subclavian arose at the beginning of the left carotid. These branches supplied the blood to the head and upper extremities.

197 COMMONWEALTH AVENUE.

NOTES ON A CASE OF INSOLATION IN AN INFANT THIRTEEN MONTHS OLD.

BY HENRI A. LAFLEUR, M.D.,

Montreal.

H. J. H., male, aged thirteen months, was brought to the Montreal General Hospital on July 2, 1894, about 1.30 P.M. I happened to be in the hospital at the time and saw the child immediately. It being a public holiday the parents had taken the child in the morning to the Island Park, just opposite the city, intending to spend the day there. The day was intensely hot and sultry, the thermometer registering over 90° during the middle hours of the day. The child had been well during the preceding days, with the exception of a slight diarrhœa. While in the park he was taken suddenly ill, and had several convulsions. The parents becoming alarmed, removed him at once to the hospital.

When seen on admission he had been insensible for an hour and a half, and had had several convulsions. The child was found lying on the examining table quite insensible; the skin was very pale, the lips, ears and hands were cyanosed; the pupils widely dilated and insensible to light. There was conjugate deviation of the eye-balls to the left. The axillary temperature was 105° F., the pulse thready and too rapid to be counted, the respiration 50, gasping and shallow. Apparently the child had but a very short time to live. He was immediately carried to the children's ward, and given a hypodermic injection of twenty minims of ether, while the rectal temperature was being taken. This was found to be 107°. Meanwhile a small bath was procured, the water being reduced to 54° F. by means of ice. The child was placed in it, a cold wet towel was wrapped round its head, and vigorous frictions of the body and extremities were carried on, with frequent affusions of ice water to the head. At the end of five minutes the rectal temperature was again taken and found to be 108° F. The same measures were carried on for fifteen minutes, and at the end of that time the rectal temperature was 101° F.

The skin was now bright red, the cyanosis had disappeared,

the pulse was about 100, and the respiration easy. The child was removed from the bath and put to bed. In half an hour after the bath the rectal temperature was 98⅘°. At 2.10 P.M., it was 95⅘°, and the patient was resting quietly, though still insensible. An enema of beef-tea and brandy was administered after the bath.

Directions were given that a bath at 80° F. should be given if the temperature rose to 102⅘° F. Such a bath was given at 5.30 P.M., and again at 10 P.M. After the third bath (10 P.M.) the child seemed to take notice of objects around him. He recognized his mother and cried a little.

July 3d, 3.55 A.M.—Temperature 103° F. A bath at 80° F. was given, lasting ten minutes. During the morning there were two small loose motions, of a greenish color. Feeding by the mouth was begun. During the afternoon there was slight cough; there were a few crepitations but no dullness at the left base. A final bath was required at 3.45 P.M. The child slept well during the night and appeared quite convalescent the next morning.

There were no further developments in the case; the temperature was still from two to three degrees above the normal, but subsided gradually during the next two days, and the child was discharged well on July 11th. I saw him some nine months later—when he appeared quite well, and according to the statement of his parents, seemed to have as much intelligence as other children of his age.

I am indebted to Dr. Bazin, one of the resident physicians of the Montreal General Hospital, for his careful notes of the case, and the unremitting attention he gave to it. The case is of interest chiefly on account of the age of the patient, and as an illustration of the happy effect of timely and energetic hydrotherapy.

58 UNIVERSITY STREET.

DISCUSSION.

DR. J. HENRY FRUITNIGHT.—I can recite a parallel case with the same result. The child was taken out one afternoon and the nurse drew the coverings tightly about the child, thus interfering with the evaporation of the body heat. The child became ill and I saw it about two hours afterward when the tempera-

ture was 103°. Two hours later the temperature was 105°, and an hour after that 107°. The condition was undoubtedly one of thermic fever. The child was delirious but had no convulsions; there were twitchings and death seemed imminent. The child was placed in a tepid bath and water from melting ice was poured over the head and body, friction being constantly used while the child was in the tub. The bath was reduced to a temperature of 65° and in this the child was kept for half an hour. It was then put to bed in a cold wet pack for one hour, when the bath was again given. The temperature was thus reduced to 102° in two hours, and by the evening of the following day the child had practically recovered. It was only by the use of ice and cold bath that the child was saved.

DR. AUGUST SEIBERT.—In a number of cases of hyperpyrexia in infants and young children I have found that the irrigation of the colon with cold water reduced the temperature of the whole body in a much shorter time than even the cold bath does. It is best to begin with tepid water, and then cool quickly within five minutes, while keeping up the irrigation for fifteen to twenty minutes. It is a very simple remedy that can be applied very quickly in nearly all cases of hyperpyrexia where there is no ice, in tenement-houses for instance, and I have only seen very happy results from it. The only case of typical insolation I have seen in an infant was years ago. The child had a temperature of 107¾° F. We succeeded in reducing the temperature quickly enough, but the child died.

In another case of hyperpyrexia in the infant son of a well-known physician in New York, the temperature rose to 107.5°, and was due to fermenting milk in the stomach. After emptying and washing out that organ the temperature fell to 103° within thirty minutes, without further remedies or the cold bath.

DR. J. P. CROZER GRIFFITH.—Philadelphia is one of the hottest summer cities of our northern seaboard, and we have at times typical cases of insolation occurring in infants. I do not refer to severe diarrhœas and similar affections from which children die, undoubtedly as the result of extreme heat, but to conditions of coma or convulsions and grave hyperpyrexia. In the latter part of June, 1894, the weather was extremely hot and in the course of five days six cases of insolation were brought into the Children's Hospital. All had slight diarrhœa and were unconscious, and most of them had convulsions. I regret that I am unable to present the full details with temperature charts. The children were all treated with the ice bath and some were placed in it in the ambulance before they reached the hospital. The ages of the children in months was as follows: 10, 10, 6, 23, 6, 23. The temperatures ranged from 106° upward. The highest temperature was 110° in a child of ten months. This lived twelve hours. Under the influence of the

ice bath the temperature fell to 98°, and the child then had to have a hot mustard bath as collapse threatened. The temperature again rose but not alarmingly, but the child died. The highest temperature followed by recovery was 107°, in a child of 23 months. This child was brought into the hospital apparently dead, and the nurse and attending physician worked over it for hours in trying to keep it alive. Two of the patients were twins, six months old, both of whom recovered.

DR. S. S. ADAMS.—In the past two or three years I have seen eight or ten cases of sunstroke in children under nine months, in which the chief symptom was rapid rise of temperature in a perfectly healthy, normal child, that was digesting his food perfectly, and was gaining in weight each week. The temperature would suddenly rise to 105° to 107° F., and the child would soon die from collapse. During the 18th and 19th of April last, a hot wave visited Washington. The maximum temperature on the 18th was 93°, the minimum 63°, the range being 30°. The greatest range during the week was 43°. During the 18th the temperature was 20° above the normal for sixteen hours. The velocity of the wind was at its minimum for the month, and the humidity was at its greatest that day, so that all the conditions for depression were present. On the 19th the highest temperature was 93°, the range was 20°, and the other conditions were similar to the previous day. Two children, under two months, in apparent health, were suddenly seized with high temperature and died. The necropsies were carefully conducted, and we were perfectly satisfied that the diagnosis of insolation was the proper one, as nothing could be found to account for death.

DR. A. H. WENTWORTH.—There is danger of confounding insolation with gastro-intestinal toxæmia. I have seen several cases of toxæmia which presented symptoms very similar to those of insolation. They die with very high temperature, collapse, and cyanosis. The amount of diarrhœa may not be excessive. Even the autopsy may show the cause of death with some uncertainty. I would like to ask if toxæmia was excluded by Dr. Lafleur in the case reported.

DR. HENRI LAFLEUR.—I am not prepared to say with absolute certainty that the case was not one of toxæmia, but I believe that it was a case of insolation.

THE OCCURRENCE OF INFLUENZA IN CHILDREN, WITH A REPORT OF LOCAL EPIDEMICS.

BY FLOYD M. CRANDALL, M.D.,

Adjunct Professor of Pediatrics, New York Polyclinic; Visiting Physician, Children's Hospital and Infants' Hospital, New York.

When epidemic influenza appeared in this country, the history of its behavior in the past led to the belief that after running an epidemic course for two or three seasons it would disappear. In view of that history, the belief was not ill-founded that the present generation of medical men would soon find but little interest in the disease except as a reminiscence. Year after year, however, has passed, each bringing its epidemic more or less severe, and presenting characteristics more or less peculiar. The belief now, therefore, is not unreasonable that the disease has become endemic, and that it may continue to appear from time to time for an indefinite period. Although it was discussed in this Society a year ago, I have ventured to present a few observations made during the past winter.

The disease, in my own experience, while it presented some rather peculiar features, and was rather prone to be followed by pneumonia of a not severe type, did not, as a rule, run a very serious course, nor were the complications particularly troublesome. It was not as general in its distribution as in some previous years. It invaded a comparatively small number of the families whom I attend, but when it did gain an entrance, it was prone to result in a decided family epidemic. In three instances this characteristic was very marked.

In the family of Mr. K. a waitress was taken ill on December 31st with typical symptoms of influenza (coryza, sore throat, fever, muscular pains, and prostration). Examination of the throat revealed the signs of acute follicular tonsillitis. On January 3d the cook was attacked in the same manner. The throat symptoms in this case were the most intense I have ever seen. In addition to the typical follicular spots, the whole pharynx, uvula, and pillars of the fauces were a mass of swollen œdematous tissue, apparently completely denuded of epithelium. Cultures

showed only streptococci. On January 5th a boy of twelve was seized with the symptoms of grippe. His throat was red and inflamed, but there was no exudate. On January 8th the father was suddenly seized with marked symptoms of grippe, the throat in his case being inflamed, but without an exudate. At about the same time a boy of nine years and a little girl of five years developed rather mild symptoms of grippe, neither showing any exudate in the throat. On January 10th the governess, a woman of about fifty, was suddenly seized with similar symptoms. During the previous week she had been in constant attendance on the cook and waitress, whose throats had been in such bad condition. Although the temperature ranged very high, and the patient was seriously ill, and the throat was much inflamed, no exudate appeared. Thus seven members of a family of nine were seized with the disease within ten days. The two to escape were the mother and a baby of two years. This baby had had broncho-pneumonia twice, once at four months and once at thirteen months, and seemed especially susceptible to catarrhal influences. He was fully exposed to influenza. The reasons for his escaping the disease are to me a mystery.

On January 17th a young girl of fifteen in the family of Mr. C. was taken ill with a disease which seemed to me to present all the characteristics of follicular tonsillitis. In quick succession the grandmother, the mother, a servant, a girl of seven, and a boy of five, developed the symptoms of grippe. I am unable to give the exact dates, except that it appeared in the above order. Although distinctive and characteristic, the disease was not severe, and no exudate appeared in the throat of either patient. A baby of thirteen months was the last to be attacked. The rational symptoms were the same as in the other cases, but in addition several round, white spots appeared on each tonsil. The disease in all cases ran a rather short and sharp course, but recovery was complete.

In the family of Mr. D. seven cases occurred, the father, the mother, and a servant being the only ones to escape. The grandmother was the first to become ill, on January 8th. On January 11th a girl of six years developed characteristic symptoms of grippe with sudden onset. On the 13th, a boy of four years developed the same symptoms. On the 14th, a girl of twenty-one months, then convalescing from broncho-pneumonia, became feverish, and developed all the symptoms of influenza.

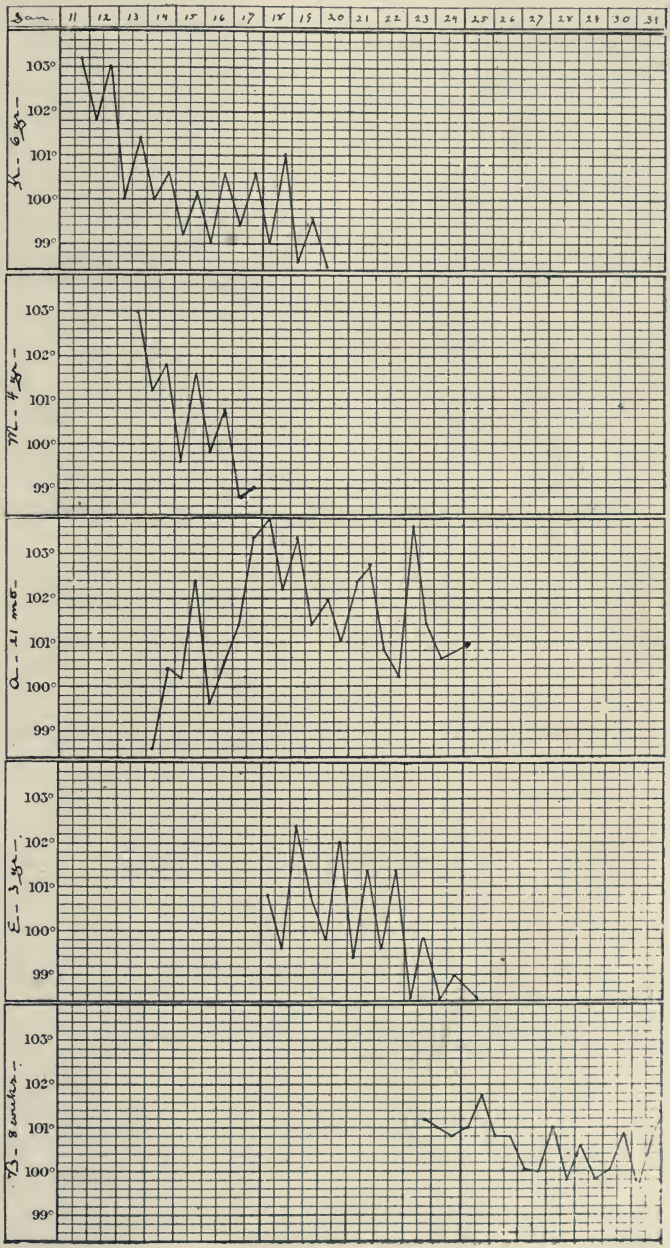


CHART SHOWING OCCURRENCE OF GRIPPE AND TEMPERATURE RANGE IN FIVE CHILDREN OF THE SAME FAMILY.

The signs of pneumonia, which had almost disappeared from the chest, reappeared, and the child went on to a second very prolonged and serious attack of pneumonia. On the same day the nurse in attendance on this child was found to have high fever, severe coryza, sore throat, and muscular pains. Four days later a little girl of three years developed characteristic symptoms, and four days after this the baby, eight weeks old, who had been thoroughly exposed for at least twelve days, also became ill. In one case of this series of seven there were the signs of acute follicular tonsillitis, the throat in the remaining cases being excessively red, but without an exudate. Of all the patients the nurse suffered from the most marked coryza, though all showed more or less suffusion of the eyes. The baby of eight weeks, although the temperature did not range high, was very ill. The skin assumed a pale and leaden look. The throat was intensely red, and so sore that the child almost totally refused to nurse for three days. It was sometimes restless, and was for a time in a semi-stupor. Its voice was husky, and it cried in a faint and wailing tone. It continued ill for ten days, and did not regain its natural condition for at least ten days longer. The accompanying temperature charts show the range of temperature in this series of cases.

Writers have differed considerably in their opinions regarding the prevalence of influenza in children. This is probably explained by the fact that the frequency of its occurrence differs considerably in different epidemics. Dr. Earle especially noted the fact that children seem to be as susceptible to the disease during some epidemics as adults, while in others the children largely escape. My experience seems to show that in the epidemic of last winter children were less susceptible to the disease than were adults. They succumbed less quickly, the occurrence of the disease in several cases being in the exact order of age, infants either escaping or being the last member of the family to be attacked. This was demonstrated in other instances than those reported.

As regards clinical peculiarities, my experience was as follows:

The rational symptoms exhibited by different patients in the same family were fairly uniform, but I was struck by the fact that in several cases the signs of typical acute follicular tonsillitis

would be present in the throat, while other cases presenting identical symptoms were entirely free from such exudate.

Except in two instances, young children did not seem to be more ill or suffer more seriously than did adults or older children. All appeared to be about equally wretched.

Soreness of the throat in infants by interfering with nursing, proved to be a rather serious symptom in several instances.

The disease expended itself chiefly on the mucous membranes of the throat and bronchial tubes. Coryza was not as pronounced as in some former epidemics.

Intense pain through the upper and anterior portion of the chest, with a feeling of tightness, was a very constant symptom, and was apparently present in very small children.

Violent spasmodic cough, usually worse at night, persisting after the acute symptoms had disappeared, and proving very rebellious to treatment, was frequent.

The abdominal symptoms so frequent during some epidemics were not of common occurrence. Impaired appetite and diminished digestive power were troublesome and stubborn symptoms in case of several young children.

After-results were not so serious, nor did they persist for so long a time as in some epidemics.

On the whole, children did not show symptoms markedly different from those presented by adults.

113 WEST NINETY-FIFTH STREET.

DISCUSSION.

DR. AUGUST SEIBERT.—I had occasion last fall to attend three of my own children with influenza with remarkable high temperature. A little girl of six years came from school complaining of feeling ill. The temperature was found to be 106¾° F. There was acute nasal catarrh and conjunctivitis. She was practically well three days later. Two younger children, one ten months old, the other two years, began the attack with a temperature of 105½° and 106¾° respectively. All recovered, although the infant developed broncho-pneumonia. Very high temperatures are easily overlooked in young children, especially in the beginning of an attack of influenza.

SARCOMA OF THE THYMUS AND BRONCHIAL GLANDS.

BY GEO. N. ACKER, M.D.,

Physician to the Children's Hospital, Washington, D. C.

Minnie S., white, aged seven years; was sent to the Children's Hospital, February 8, 1896, by the Board of Children's Guardians.

The brother and sister have hereditary syphilis.

For the last five weeks the child has been dull and quiet. She has lost flesh, and has had no appetite. During this time a large swelling developed on the left side of her neck,

She is poorly nourished and anæmic. The left side to the waist, and the left side of the face and head, are very œdematous. The left arm is enormously swollen. There are numerous mucous râles in both lungs. The impulse of the heart is downward, and to the right. The child is very restless and complains of thirst. The urine is normal. Has had two loose greenish undigested stools since admission into the hospital.

At 9 P.M., temperature, 101.5°; pulse, 140; respiration, 66. She was put on a milk diet, and Syrup. Acidi Hydriodici (twenty minims), every four hours was ordered.

February 9th. The right side of face and right arm began to swell to-day. She grew weaker during the day, and failed to respond to stimulants. At 11 P.M. she passed quietly away.

At 9 A.M., temperature, 102.2°; pulse, 135; respiration, 60. At 9 P.M., temperature, 103.2°; pulse, 150; respiration, 60.

Post-mortem Examination.—Body well nourished. Rigor mortis marked. Much adipose tissue. The left mammary region is infiltrated with fatty tissue to the extent of two inches. The left axillary space contains a large growth which extends over the clavicle, pressing on the left sub-clavian vein. The right axilla presents a somewhat smaller growth. A large, soft, lobulated tumor lies in superior mediastinum, surrounding the trachea, and great vessels, and extending to, and involving part of the upper lobe of the left lung. The lungs show some old and recent pleurisy. Both are œdematous. The heart is normal. The liver is pale. The kidneys are somewhat enlarged and pale.

Microscopical examination of the growth shows it to be a large round cell sarcoma.

913 SIXTEENTH STREET.

ON THE CLASSIFICATION OF THE TICS OR HABIT MOVEMENTS.

BY WILLIAM OSLER, M.D.,

Professor of Medicine, Johns Hopkins University.

Linnæus "who found botany a chaos, and left it a cosmos," is said to have had the courage to write a treatise on the *Genera Morborum*. While the present condition of the classification of disease can hardly be called chaotic, yet order and system are necessarily lacking in the absence of a fuller knowledge than we now possess of the mutual relations of various disorders. There are, indeed, insuperable difficulties in the way of any broad systematic arrangement upon a basis either etiological or anatomical. Meanwhile, we jog along in an aimless fashion, ticketing the maladies according to their seeming similarity, adding daily to the existing complexity, and waiting for some twentieth century Linnæus with a genius for classification.

Nowhere is the confusion more evident than in the classification of diseases of the nervous system, particularly in the disorders characterized by abnormal movements. Even in an affection so well studied as chorea, it is very difficult to make a classification that will meet with universal approval. How can it be otherwise? We are by no means unanimous, either as to the nature of chorea minor, or as to the relations of other motor affections to it; for example, of the chronic progressive chorea of Huntingdon, which is regarded by most writers, and I think correctly, as separate and distinct from Sydenham's chorea; while Charcot and his pupils hold that it is only a variety.

Important studies have been made of late years upon the group of muscular disorders which have been described as

pseudo-choreas or habit spasms, and, in extending to them the name of Tic, the Salpetrière school has developed a nomenclature and division somewhat confusing to Anglo-American ears. With our notions of the word tic applied to either the mimic spasm of the facial nerve—tic non-douloureux, or to neuralgia of the trigeminus—tic douloureux, it is a novel extension to hear such phrases as *la nèvrose tiqueuse, la maladie des tics convulsif, ticquer, tic psychique* and *tics coördonnés.* With the exception of Dana, recent authors of works on diseases of the nervous system, have not adopted either the nomenclature or the division recognized by the Salpetrière school. It has certainly advantages, particularly in enabling us to separate a number of the spasmodic affections of children from chorea minor. The disadvantage lies in the use of a name already attached to two well-known and totally different affections, the spasm of the facial nerve and the neuralgia of the trigeminus. Still there need be no confusion in reality. The facial tic, like the spinal accessory and the hypoglossus spasms, differs entirely from the habit spasm, in as much as the convulsive seizures are situated within the domain of a definite nerve, and there is generally some lesion, central or peripheral.

The employment of the word with a significance different from that to which we are accustomed, is compensated for by the advantage of placing under it a group of allied affections which pass insensibly into one another, from a simple, habitual, conscious spasm of the facial muscles, to complex coördinated movements with marked psychical features, or to habit phenomena purely psychical. The distinguishing factor in the entire group is the habit or repetition, whether of motion or of idea, which, while influenced or controlled to some extent by the will, occurs in response to a sort of impulsion in the case of muscular movements, and in the case of imperative ideas as a sort of obsession.

The following is the classification of the tics adopted by recent French writers.*

1. Simple tic or habit spasm. These are the common cases of spasmodic movement, chiefly of the facial muscles, which are, to a certain extent, under the control of the will. In very many of these cases the affection seems to begin as a childish trick.

* See particularly Noir *Étude sur les Tics. Paris*, 1893.

The muscles of the face and of the neck are most commonly involved, then those of the shoulder girdle and arms, less frequently those of the legs. The cases are very often confounded with chorea minor, from which they are distinguished by the much greater brusqueness of the movements, which have a lightning-like rapidity. They are also more or less systematic, limited to certain muscle groups, as of the face and neck, or of the shoulder girdle muscles and platysma, or the muscles of the thigh. In many of the cases there are sources of irritation, such as adenoids or errors of refraction, relief of which may be followed rapidly by cure. In addition to these localized forms, here may be also grouped as a generalized tic or habit spasm, those interesting cases in which there are sudden electric-like jerkings of the muscles of the trunk and extremities, making the patient start for an instant, but which pass away with great rapidity. Both children and adults are affected, and Henoch has described the condition as electric chorea—not a very suitable name, since this has been applied to the totally different affection known as Dubini's disease, met with in Lombardy. The condition may persist for many years, and in my monograph, *On Chorea and Choreiform Affections*, I have recorded several interesting cases. These habit spasms, whether local or general, often resist all methods of treatment, and, while never dangerous, are extremely annoying and a source of great worry to the unfortunate subjects. They should not be confounded with chorea, nor should that term be applied to them, but they are best designated either by the term habit spasm or simple tic.

2. Tics with super-added psychical phenomena, *Maladie de la Tic Convulsif* or Gilles de la Tourette's Disease. The study of these cases by Gilles de la Tourette, and by others of Charcot's pupils, really led to the extension of our knowledge on the subject of these curious affections. In this form, in addition to the ordinary motor disturbances of simple tic or habit spasm, there are explosive utterances and cries, and imperative ideas. Of these the anomalies of expression are the most interesting. There may be simply explosive exclamations, which are most frequent, or obscene words may be spoken, usually at the time of the movement—the so-called coprolalia; or a word heard is repeated a number of times, or some one word is said over and over again, for which habit the term echolalia has been invented. In other instances actions are mimicked—echokinesia, and it seems

probable that the jumping disease of Maine, the Latah of the Malays, and the Myriachit of Siberia, all of which affections are characterized by this condition of *echomatism*, come really under the category of the *tic convulsif* of Gilles de la Tourette. But the most interesting, as well as the most distressing feature of this variety of tic is the remarkable mental state, usually some form of obsession or imperative idea. They are very varied; many of them are the modifications of the various *phobias*, for which so many names have been invented, agoraphobia, topophobia, claustrophobia, haphephobia, etc. Or there may be the curious conditions of onomatomania, or of arithmomania, or in other instances the mental state is that of *folie du doute* or the *délire du toucher;* interesting cases of these I have given in the monograph already referred to.

3. Complex, coördinated tics. By far the best account of these is given in the exhaustive article on Tics by Noir, writing from Bourneville's clinic. Many are forms of habit movements which differ, however, from the simple tic in the more complex character of the action performed, which may be one of every-day life, but which is repeated without obvious cause, and which, in most instances, can be controlled by an effort of the will. Some of the more complex movements do not differ at all from ordinary tricks, or the complex movements may occur in connection with ordinary habit spasm, as in a child who always before taking anything in the hand, first smelt and then blew upon it; or a boy with facial tic, who had the habit of biting the middle finger, and at the same time pressing the point of the nose with the index finger. Some such tricks in children as head-nodding, head-swaying and head-banging, come in this category. In feeble-minded children one sees a very extended series of these complex coördinated movements, of which a very excellent account has been given by Noir, particularly the balancing, the jumping, the rotation of the head and the rhythmical beating of the head or of the chest with the fist. The movements are usually rhythmical in character. Sometimes a series of actions is performed from time to time in orderly sequence, such as stooping from the chair, lying prone upon the floor, raising the hands above the head, etc.

And, lastly in this group, come most appropriately those extraordinary bizarre movements, which may be repeated from time to time for a series of years, sometimes in association with

explosive utterances, or with imperative ideas, as the extraordinary case of pendulum spasm reported some years ago by Mitchell.

4. Tic psychique. An imperative idea is the psychical equivalent of, and has an origin similar to, the motor tic. The *idée fixe* impelling the victim to touch a certain object, or causing him to be haunted by a dread of the use of certain words, or making him count so many numbers before he can do a certain act, is the counterpart of the irresistible musculation which leads to the constant repetition of one of the many acts which we have been considering. The two processes are as we have seen, often though not necessarily associated, and in some of the subjects of imperative ideas the motor features are marked. In any of the cases the psychic tic is as harmless as are the slighter forms of the motor variety. Dr. Hack Tuke has called attention in a valuable paper to the trifling character of a large number of the imperative ideas. They may be present for years without delusions, recurring automatically, often proving a source of worry, but rarely becoming more serious than other of the many every-day ideas which from habit we entertain.

The following works may be consulted: Gilles de la Tourette's *Archives de Neurologie*, 1885; Guinon, *Dict. Encyclopedique, Article Tic*, 1887; Charcot, *Leçons du Mardi*, 1887-88, 1888-89; and in his last lectures, 1893, edited by Guinon; Noir, *Étude sur les Tic*, 1893; D. Hack Tuke, *Brain, Part Second*, 1894; Dana, *Text-book of Nervous Diseases, Second Edition*, 1894; Osler, *Chorea and Choreiform Affections*, 1894.

ELIMINATION AS AN ETIOLOGICAL FACTOR OF DISEASES OF THE ALIMENTARY CANAL.

BY F. FORCHHEIMER, M.D.,

Professor of the Diseases of Children, Medical College of Ohio, University of Cincinnati, Cincinnati.

The process to which I wish to call attention is one that is universally accepted at the present day, so that from a physiological standpoint, there is no doubt of its existence. As to its practical application, it has seemed to me that it is very manifold and not limited to the mucous membrane of the alimentary canal, but a function of all mucous membranes. It is not within the scope of this paper to discuss this general applicability of the principle; it shall be my object to point out a few instances in the various sections of the alimentary canal where this principle becomes operative.

The term elimination has been chosen because the process, strictly speaking, is neither one of secretion nor of excretion, although both of these terms have been applied to it. During the process of secretion a mucous membrane may eliminate from the circulation substances of various kinds, which substances may be secretory or excretory, or neither. The substances that we refer to are certainly not destined to perform any physiological functions in the body, therefore, not secretory as a rule, they are not waste products; therefore, not excretory, it will be convenient to use the term eliminative for them. The greater the amount of secretion, the greater the amount of elimination; so that, as far as our researches go, it has been shown that the latter process goes on with least activity in the large intestine. It would almost seem that the actual amount of eliminative function had something to do with the determination of the frequency of pathological processes in the mouth, the stomach, the small intestine, and the rectum. It seems, furthermore, to be true that elimination takes place most actively in those sections of the alimentary canal in which absorption is

least developed. This fact seems to depend for its explanation upon the relation of secretion to absorption.

The substances that have been found to be eliminated are of very varied nature; metals, salts, alkaloids, ptomaines, leucomaines, coloring matter, all may be eliminated by the mucous membranes of the alimentary canal.

The question how any given substance, found in the economy, may be eliminated by a mucous membrane, must still be left unanswered. It is highly probable that, as in the general metabolism, the one substance is a respiratory eliminative, the other a digestive eliminative, the third an excretory eliminative, because it is eliminated especially by the mucous membrane of one or the other apparatus. In a series of experiments that I have been engaged upon, I have tried to determine something about the mechanism of elimination. The substance to be eliminated was a non-toxic coloring matter; as far as the alimentary canal was concerned, it mattered not whether this coloring matter was injected into the arteries, the veins or the lymphatic spaces of a rabbit, it seemed to appear in the same quantities in the lumen of the intestine. So that the conclusion could be fairly drawn, that, wherever the substance to be eliminated was formed, it could be eliminated by the intestine. On account of technical imperfections of the method, it was impossible to determine the tissues that were involved in elimination. It is highly probable however, that substances vary in this respect, as has been suggested before. For our present contention we must disregard the origin, and, to a certain extent, the nature of the bodies to be eliminated; therefore, that most interesting process first described by Brown Sequard, and elaborated by d'Arsonaval and others, internal secretion.

Depending upon the nature of the body eliminated, the diseased process must of necessity vary; an inflammation of different degrees of intensity, a functional disturbance, or no disturbance at all. Again, remote disturbances may be ascribed to this process if it be remembered that these eliminated substances may again be absorbed, that while this may not injure the mucous membrane primarily, they may do harm to the cells of other tissues, or effect chemical changes of great importance, being changed so that when they again come back to any mucous membrane, they secondarily may become capable of producing damage. If we then take this principle of elimination

into consideration, we have before us a nice problem involving the question of secretion, auto-infection, excretion and, secondarily, the whole metabolism of the body.

As far as the mucous membrane is concerned, a substance of this sort can produce a lesion or disturbance either upon its first or subsequent appearance upon the mucous membrane, and *vice versa*. Indeed, we know of substances which are toxic when first eliminated, become absorbed, then changed in the economy to be returned as harmless bodies. See the relation of ammonium carbamate to urea.

Turning now to the various sections of the alimentary canal, we will find very many examples of disease due entirely to eliminative processes. The mouth has very little activity as far as digestion or absorption is concerned, therefore we would expect elimination to be of the utmost importance in the production of disease in this locality. As far as experimental evidence goes, we know that many substances are eliminated by the glands of the mouth; metals, salts, and organic products. There can be no doubt of the fact that the condition that precedes some forms of stomatitis (stomatitis hyphomycetica, stomatitis ulcerosa) is due to an irritation following the elimination of substances that have as yet eluded detection. It is a well-established fact that stomatitis is frequently associated with gastro-intestinal disturbances, and it has seemed very improbable that an inflammation could extend from the small intestine, by some unknown connecting substance, directly to the mouth and then produce an inflammation. Bacteriological research has not been able to help us much in the determination of the etiological factors for stomatitis, as the bacterial forms were found to be mostly the ordinary ones of inflammation. The fact that the soil must be properly prepared before the bacterial activity can take place, has been pointed out by me in my book on "Diseases of the Mouth." Stomatitis ulcerosa is a disease whose cause is accepted as being the existence of a proper soil upon which some irritant or irritants produce inflammation of a peculiar nature (necrobiotic). If we analyze these conditions we will find three factors: the presence of teeth (mechanical), the elimination of certain substances, and lastly, though possibly not essential, the presence of certain lower forms of life. Eliminative inflammation is accepted in this form of disease for those cases due to mercurial, arsenical, iodine, and lead poisoning. Another

chemical disease, scurvy, can be safely added to this list, and when chemistry advances still further, there can be no doubt but that all forms of stomatitis ulcerosa will be found to be primarily an eliminative inflammation. In this connection I wish to draw attention to the constitutional origin of that form of disease that resembles stomatitis ulcerosa, but chronic in nature, Riggs' disease or pyorrhœa alveolaris. It has long been conceded by dentists that there is something more than a local cause for this disease, and their futile attempts at treatment must be ascribed to the fact that they were not in a position to treat anything more than the local manifestations. Now that the dentist is calling on the physician to determine the general cause, the results of treatment are becoming very promising. But what do we find as general causes for this disease? Only such as produce certain foreign chemical substances, or are produced by such substances circulating in the blood, and the mechanism is simply that of elimination as in stomatitis ulcerosa. Of these substances uric acid seems to be the principal offender. It has been found by Boucheron in the saliva, and while I am far from accepting much that has been written upon uric acid as a cause of disease, it can be safely admitted that uric acid, locally, does produce inflammations. The various forms of anæmia, malaria, diabetes, also act causitively, and that in each of these diseases chemical substances are produced, need not be alluded to more extensively. Stomatitis herpetica seems to be conclusively determined upon as an eliminative disease by the interesting observations of Friedrich, and, parenthetically, it may be stated that the nomenclature of our society has been fully vindicated in calling stomatitis aphthosa a herpes. Friedrich (Berl. Klin. Woch. No. 49 and 50) in the course of some experiments made with the proteins and toxalbumins of streptococcus and the bacillus prodigiosus as used by Coley, was able to produce herpes facialis in seven cases, in two of which the herpes also appeared within the mouth.

The accepted functions of the mucous membrane of the stomach can be arranged in the following order as to importance: secretion, elimination, absorption. It is difficult to rate the eliminative function as to quantity, but it will probably be found to be the second one in importance. It would carry us too far if we were to review the history of the change in opinion among physiologists concerning the functions of the stomach in the

human being. It will be necessary, however, to give a few facts pointing to the importance of considering the stomach as an eliminating organ, which, as yet, have not been taken up by the text-book on physiology. It has been found that a great variety of substances is eliminated by the stomach, discovered first in the natural course of repeated chemical examinations according to the methods of Leube, Ewald, and others, then worked out as the result of preconceived notions concerning this function of the stomach. In the latter way, and possibly to serve as a paradigm, I found some remarkable things concerning the elimination of arsenic by the stomach. Arsenic is eliminated by the stomach in large quantities, is then absorbed, and again eliminated, and this continues for weeks in acute cases of arsenical poisoning, so that, even by means of the coarsest tests for arsenic, this substance can be detected in washings from the stomach. In one way or another the following substances have been found as eliminated in the stomach: manganese (Cahn), arsenic (myself), uric acid (Boucheron), xanthin (Rachford), a ptomaine with the action of strychnine (Brieger), morphia and the cholera toxin (Alt), organic alkaloids in auto-intoxication and absorption-sepsis (Albu). The mucous membrane of the stomach seems to react in a peculiar manner; thus while manganese and arsenic are eliminated, as yet I have failed to detect lead in the stomach contents of patients with lead poisoning. On the other hand, it seems to be able to separate organic compounds of widely different natures, and herein lies the importance of recognizing this function of the stomach. The stomach tube has been accepted quite universally by the profession. It has been used indiscriminately in any condition which seemed to warrant the supposition of change in gastric digestion, and most commonly with excellent results. The most important function of lavage has been overlooked, however, viz., that of removing something from the stomach which has been secreted into it and eliminated from the general circulation. Lavage in cholera infantum offers most inconstant results—sometimes most brilliant, more so than by any other method of treatment, and that occurs when both the cause of the disease (bad milk, bacterial contents) and its results (eliminated substances) are removed from the stomach. This would occur early in the disease: or the result may be excellent in those cases in which the origin of the disease is neither chemical nor bacterial, *i.e.*, as far as the primary conttens

of the gastro-intestinal cause is concerned, but due to elimination into the latter as the result of some process generally metabolic. The mechanism of the production of cholera infantum, as the result of great heat, concerning whose existence there has never been any doubt in my own mind, can be most satisfactorily explained by taking elimination into consideration. Lastly, there are those cases of cholera infantum that are only temporarily benefited as far as vomiting, general condition, and general results are concerned. These are the cases in which the cause is not reached, and the lavage acts only palliatively, for the poison continues to act continuously.

A great many acute gastric conditions concomitant with other diseases will undoubtedly be explained on the principle of elimination. The vomiting of the acute infectious diseases must undoubtedly be brought partially under this heading. The gastric disturbances of trouble of the lower sections of the bowels, the vomiting of constipated children, for instance, which is frequently considered mechanical, is due purely to auto-infection and an attempt at elimination by the stomach.

It would be going too far to do more than suggest that a great many of the chronic or sub-acute gastric disturbances in children are simply due to the same process, and not to disturbances in secretion of gastric juice, pepsin or what not. It is for this reason that direct treatment of the stomach has offered such good results, because in nearly all instances, lavage is an essential part of the treatment. Clinicians know that the gastro-intestinal troubles of rickets, tuberculosis, so-called gout, must be treated by applying remedies to the cause. At the same time most excellent temporary results are obtained by continuous lavage.

In the small intestine the conditions are most unfavorable for study and conclusion for the present. It must be remembered that the physiology of the small intestine in man, is, comparatively, an unopened book; with the exception of the observations in Busch's classical case, comparatively little is known positively. In addition, the normal processes are so complex, the juices and contents are so many, bile, succus entericus, pancreatic juice, the chyme, that conclusions must be carefully sifted before they can be accepted. If we take the bile and see what extraneous substances has been found in it, we will see how this fluid must be eliminative par excellence. Thus, there may be found metals,

iron, copper, arsenic, salts, iodides, bromides, sulphides, sodium salicylate: of organic substances, carbolic acid, urea, turpentine, leucin and tyrosin, albumins, cane and grape sugar and other substances. The small intestine eliminates calcium and iron (Voit, Rey, Buchheim and Meyer), iodine, bromine, lithium, arsenious and boric acids, iron salts, (Landois and Sterling); manganese (Cahn). Brandenburg has found that uric acid formation has some connection with the secretory function of the intestine, and Weintraud goes so far as to state that the precursors of uric acid, which are formed from nuclein derivates, are secreted into the intestinal canal, then reabsorbed and finally changed into uric acid. In so simple an experiment as the determination of the place of elimination for Heidenhain's sulphoindigotic carmine, I was at first thwarted by the complex conditions in the small intestine. But this experiment was, primarily, for the purpose of purely theoretical considerations. Here we wish to establish the fact that conditions in the small intestine are very favorable for elimination, but that, at present, we have not a very satisfactory basis upon which we can build practical results. If we look at the great number of substances eliminated by the bile, it will be seen how important this function must be for this juice. If then we follow the rules used before, and take into consideration the great quantity of fluid secreted in the mucous membrane of the small intestine, we will also have to come to the same conclusion as to the succuss entericus. The difficulty however, lies in the isolation of substances at the place in which they are found, which, to a certain extent, is necessary to prove the position taken as to elimination. If we draw conclusions as to the importance of the function, as the result of years of accumulated therapeutical experience, it will not be difficult to establish the fact. The cures of diseases of various kinds, the relief given to uremic conditions, the treatment of certain kinds of intoxications by prolonged catharsis, all point to the importance of this function in the small intestine. It is not an uncommon practice, and frequently a very good one, to begin the treatment of an acute gastro-intestinal disorder by giving a laxative. No doubt the materies peccans is removed by this method, *i.e.*, the primary cause of the trouble, if it be a lower form of life. But we must not lose sight of the fact that lower forms of life have produced toxins or toxalbumins, or that, to begin with, this disorder is due to the introduction of some toxic-albuminous

substance, and in either case these bodies are absorbed to cause the constitutional symptoms. Now these substances are eliminated by the intestines, and a great part of the value of a dose of calomel is to be attributed, not to its being an intestinal antiseptic, or being converted into sublimate or what not, but simply that elimination is increased by stimulating the bowels to movement.

If we now come to a discussion of the large intestine as related to elimination, we will have to start with the following proposition. The principal function of the large intestine is that of absorption. Klug and Kovack claim that its absorptive power is greater than that of the stomach. It is also secretory (Heidenhain) but to a small extent. There is very little fluid secreted, the physical condition of the secretion changing rapidly to mucus as we go from the small intestine to the rectum. The conclusion that we would arrive at from these conditions is that elimination is not well developed in the large intestine, and this conclusion seems warranted if we review the etiology of the diseases that occur in the colon and rectum. But there are some conditions the result of an excess in elimination as to quantity or quality, that undoubtedly produce disease in the large intestine. The best known is that one due to intoxication. Mercurial elimination has been established as a cause for trouble in the large intestine that may become serious. It is highly probable that this principle must be taken into consideration in connection with many other pathological conditions of the large intestine, but here again it is difficult to disassociate eliminative processes from others. The lower we go in the intestinal tract the more difficult does this become, so that while in the mouth we could say, more or less positively, what was due to elimination; in the rectum we find so many factors to take into consideration, mechanical, physical, physiological and others, that further study is required before we can decide upon the nature of the process. It has seemed to me that, besides the intoxication, there were severe general conditions that acted upon the large intestine by elimination; the intestinal trouble of influenza, of malaria, of pneumonia, in some instances that of tuberculosis. I have seen several patients who had that form of large intestinal trouble known as enteritis membranosa, in all of whom the condition disappeared as the result of treatment of some tubercular condition.

In conclusion it will be seen how large a field is opened up in etiology, by taking into consideration this process of elimination. Besides the interest attached to it from a purely scientific point of view, there is the more important one of its relation to therapy. In the review of the subject, necessarily incomplete, little stress has been laid upon this aspect of it; but, none the less must we all be impressed by the feeling that, after all, the great duty of the physician is to cure his patients. Anything that can help us in this direction must be of great value, and, therapeutically, elimination has been of great service to many, even before the principle was thoroughly understood. Let us hope, more so now that it is being grasped and made the basis of tentative therapeutic measures, which, in many instances, prove successful.

THE ORTIZ, FOURTH AND SYCAMORE STREETS.

A CASE OF APPENDICITIS, FOLLOWED BY GENERAL PERITONITIS, IN A CHILD TWO AND A HALF YEARS OLD.

BY L. EMMETT HOLT, M.D.,

Professor of Pediatrics, New York Polyclinic; Attending Physician to the Babies' and to the Nursery and Child's Hospital, New York.

Attacks of appendicitis in very young children are so infrequent, that even when they occur they are likely to be overlooked, as the subjective symptoms upon which we depend so largely for diagnosis in older patients are wanting. The following case is interesting because of the obscurity of the symptoms and the age of the patient:

A. R., female, two and a half years old, was seen on April 15, 1896, in consultation with Dr. W. E. Bullard. The child had been exceptionally strong and well since early infancy. For a month before the present attack began she had been somewhat constipated, but with assistance the bowels moved every day. On the afternoon of April 12th, she was taken, three hours after a hearty dinner, with vomiting; this was of the contents of the stomach, but continued after the stomach had been emptied, and was repeated at least a dozen times during the following night, accompanied by some fever. The child did not complain of pain, and did not appear to be seriously ill. On the following morning a cathartic was given, and this was followed in the evening by a good-sized normal evacuation. She vomited occasionally through the day.

On April 14th she was very bright, and by the parents was regarded as quite recovered. She went out in her baby-carriage, and wanted to get up and run about. Toward evening, however, she developed fever, complained of indefinite pain, and vomited again once or twice. She had been kept upon a fluid diet since the beginning of the illness. She grew rapidly worse through the evening, and at 11 P. M. Dr. Bullard saw her for the first time. There was considerable prostration, quite a rapid pulse and high fever, apparently about 103° or 104°.

On the following morning, the 15th, she was no better, and was kept in bed all day for the first time. At 10 A.M. the pulse

was 110, temperature 102°. Vomiting was repeated several times during the day; now the vomited matters were of a grass-green color; previously they had been yellow.

Examination at 4 P. M. showed a stout, well-nourished child, who appeared to be quite seriously ill. The tongue was slightly coated; the mind clear. Respirations were rapid, about sixty a minute, and the nostrils dilated actively with each inspiration; she moved freely about the bed, and seemed comfortable in almost any position. Her general symptoms were strongly suggestive of pneumonia, and she had coughed a few times during the early part of the day. Examination of the lungs, however, revealed nothing, except rather rude respiration over both sides posteriorly. On account of the repetition of the vomiting, and the character of the vomited matters, some intestinal obstruction was suspected, and chloroform was administered, and the abdomen examined with great care. No tumor was discovered at any point; there was no induration in the right iliac fossa; and neither by external examination or *per rectum* was anything suggesting intussusception or appendicitis discovered, although the child was carefully examined with reference to both these conditions. There was no retraction of the thighs, and they could be moved freely in all directions. There was a slight tympanites, but the bowels had not moved since early morning. There seemed to be a slight amount of abdominal tenderness, but it was nowhere marked, and could not be localized.

On the following day she grew rapidly worse; the prostration became very marked, and the temperature fluctuated between 104° and 105.5°. Vomiting was repeated, and all vomited matters were grass-green. This, with the steadily increasing tympanites, left no doubt whatever regarding the existence of peritonitis. On the night of the 16th she grew alarmingly worse, and died on the morning of the 17th, five days after the beginning of the attack, and three days from the time when the symptoms appeared serious.

Only a partial autopsy could be obtained, and this was made under some difficulties, as the embalmer had already injected the abdominal cavity. The lungs were normal, except a moderate amount of congestion. There was no pleurisy; there was a general peritonitis, the intestines being coated quite generally with recent lymph. There was an opening in the appendix about one inch from its distal extremity, and just above this

point was a hard concretion about the size of a pea. The margins of the opening in the appendix were in a sloughy condition. There was no abscess, and no localized induration in the right iliac fossa. The liver, spleen and kidneys were normal.

Remarks.—The essential features of this case were that a previously healthy child was suddenly taken with vomiting, which was very persistent at first, but continued throughout the attack, the vomited matters being grass-green after the third day. The bowels were moderately constipated. There was no great amount of prostration until the last thirty-six hours of the illness. There was slight fever at the onset, and high fever for the last two and a half days.

At the time of my examination, upon the 15th of April, there was nothing in the face, pulse, or general condition to suggest peritonitis, the only symptoms pointing in this direction being the grass-green vomiting and the very slight amount of tympanites. From the slight cough, rapid respiration, and negative evidence obtained by a careful examination of the abdomen under chloroform, I was inclined to the diagnosis of pneumonia with abdominal symptoms. On the following day, however, peritonitis was very evident. At no time during the progress of the case were the symptoms clear enough to justify operative interference.

15 EAST FIFTY-FOURTH STREET.

DISCUSSION.

DR. WM. OSLER.—A few days ago a colored child of three or four in my ward (a convalescent for nearly three months from severe pneumonia after diphtheria), was taken ill on a Tuesday with severe pains in the upper part of the abdomen. The intensity of the pain, and the high fever, made Dr. Thayer suspicious that there was possibly a recurrence of the pneumonia. On Wednesday the fever persisted; she had vomiting, and the pains were localized in the abdomen, particularly on the right side, but there was no positive tumor. On Thursday morning, when I saw her, it was quite evident that the child had peritonitis, and I sent her down to the operating room at once. There was a diffuse peritonitis, a great deal of injection over the membranes, an acutely inflamed appendix, not perforated. There was a sero-purulent exudate, and from it, and from the appendix, cultures of the pneumococcus were obtained.

CICATRICIAL STENOSIS OF THE LARYNX IN A CHILD.

BY AUGUSTUS CAILLÉ, M.D.,

Professor of Pediatrics, New York Post-Graduate Medical School and Hospital; Visiting Physician to the German Hospital, New York.

The larynx here presented was taken from the body of a girl three and one-half years of age, who was tubed in December, 1895, for diphtheritic croup, at the Willard Parker Hospital, and who subsequently contracted measles, and was transferred to North Brother Island, still wearing the tube. During and after her illness (so it was stated), the child required constant watching, as she was liable to cough up the tube at any time, and was unable to breathe as soon as the tube was out.

I saw her on March 9, 1896, and suspecting cicatricial stenosis of the larynx, I prepared myself to perform a rapid tracheotomy, and I then took out the tube. Breathing immediately became very labored, and in less time than it takes to relate it the little patient was deeply cyanosed, and in a state of complete asphyxia. As the old tube was very much encrusted and roughened over its entire surface, I introduced another tube of the same size. The obstruction which I encountered at the entrance of the larynx was overcome without much force, and in about one minute after placing the tube into the larynx, the child had its normal pink and white color, and a contented smile. I diagnosed a cicatricial stenosis at the entrance of the larynx, and advised tracheotomy with subsequent dilation, or laryngotomy. The operation was to be done on the following day, at the Babies' Wards, Post-Graduate Hospital. In order to run no risks as regards coughing up of the tube, the child was transferred to the ward at once, and everything made ready for tracheotomy at a moment's notice. At 10 P.M. the child awoke from its sleep, and coughed up the tube. The house surgeon was in the ward at the time, and at once placed the child upon the operating table, and with a few strokes of the knife opened the trachea, but the child was dead.

By examining the specimen, you will recognize at once the cicatricial stenosis at the entrance of the larynx, probably the result of a pressure necrosis or direct injury in tubing.

753 MADISON AVENUE.

DISCUSSION.

DR. W. P. NORTHRUP.—This child was for a long time under my care at the Willard Parker Hospital. It was then suffering from adductor paralysis, the diagnosis being confirmed by Dr. O'Dwyer. As a patient, the child caused much anxiety in the hospital. It would frequently cough the tube out in the night, and the whole house would be aroused to prevent the death of the child. Dr. O'Dwyer believed that if tracheotomy were performed to get rid of the paralysis, that intubation would have to be performed to get rid of the tracheotomy tube. It is possible to make a diagnosis of adductor paralysis without the laryngoscope. The diagnosis in this case was made upon physical evidence and the behavior of the tube.

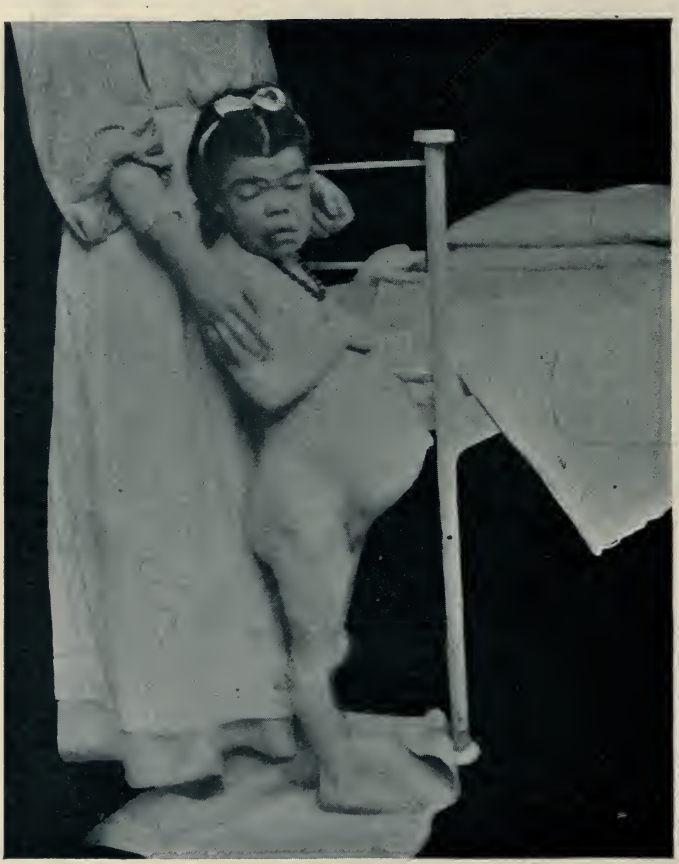
DR. A. CAILLÉ.—I wish to go on record as stating that it is impossible to make a diagnosis of adductor paralysis without the laryngoscope, and I believe that the specimen shown by me is one of cicatricial stenosis. It may have been adductor paralysis at one time, but by examining the larynx here presented, any one can see that there is a cicatricial stenosis. If I had tracheotomized the child immedately, it would be alive to-day, but I did not get the consent of the parents at the time. Tracheotomy should have been done at a much earlier period in this case.

A PREGNANT CRETIN.*

BY CHARLES W. TOWNSEND, M.D.,

Physician to Out-Patients, Massachusetts General, Children's, and Boston Lying-in Hospitals, Boston.

The accompanying photograph is of a cretin, seven months pregnant. She was born in Eastern Massachusetts, of American



CRETIN, THIRTY-EIGHT YEARS OF AGE, PREGNANT.

*Photograph shown at the meeting of the American Pediatric Society in Montreal, May 27, 1896.

parentage, and is now thirty-eight years old. She is forty-three and one half inches in height, and weighs with clothes, and in her pregnant condition, sixty-five and one-half pounds. Her catamenia began at the age of twenty-two years. She is an idiot and her small size and thickened features present the characteristic appearance of cretinism.

She had, of course, a small pelvis. Cæsarian section was performed at full term, July 26. 1896, at the Boston Lying-in Hospital by Dr. Edward Reynolds. The mother made a good recovery. The infant was fairly developed showing no signs of cretinism. It was a male weighing three pounds and ten ounces. It was very feeble and lived only four hours.

76 MARLBOROUGH STREET.

A CASE OF MULTIPLE TUMOR OF THE BRAIN.

BY HENRY DWIGHT CHAPIN, M.D.,

Professor of Diseases of Children at the New York Post-Graduate Medical School and Hospital.

Florence L., two years and a half old, was admitted to the Babies' Wards, October 24, 1895, in a condition of mild stupor, or deep sleep.

Family History.—Both parents rheumatic; seven other children who had never had serious illness. There was no tubercular history in the family.

Previous History.—Measles was the only illness before the present attack. The child had been well up to four months ago, when she suffered from a severe attack of diarrhœa, lasting two weeks. Soon after this she grew drowsy, sleeping much of the time, and has never since shown any interest in surrounding objects. When spoken to, she nods her head, but does not reply. No vomiting and no convulsions. She takes only soft diet. Bowels inclined to constipation. The eyes are closed all the time, and it is by apparent effort that she can open them. There has never been complaint of any pain.

Physical Examination.—General development good; heart and abdomen negative; slight dullness on percussion at apex of left lung—otherwise negative. *Reflexes.*—Knee-jerk and ankle clonus absent. *Sensation.*—Tactile, heat and pain normal. Hearing and smell are good, and taste is apparently so. The sight is doubtful. The child can lift the eyelids but hardly enough to see. The pupils are regular and react to light, but are slightly dilated. When the child is raised, she hangs over in a limp condition, the head dropping forward or backward according to the direction in which the body is bent. When the child lies on her back, if either arm or leg is raised, it remains in that position for about half a minute, and then slowly drops. When asked where she feels sick, she puts her hand on the back of her neck. Urine normal. Temperature, 99° F.; pulse, 128; respiration, 30; weight, 16 pounds, 3 ounces.

ABSTRACT OF CLINICAL HISTORY WHILE IN THE HOSPITAL.

October 25th. In constant stupor, but takes soft food well. She can be temporarily aroused by shaking.

October 29th. Sat up in bed to-day with body and head bent forward. She cries out occasionally as if in pain, and when raised, falls forward and screams. No stool in the past sixty hours.

November 1st. Examination of the eyes by Dr. Davis. The fundus of both eyes normal; slight paleness of disks on temporal sides, but this is not pathological; some nystagmus noted.

November 3d. Eyes still closed most of the time. Put upon iodide of potash, ten grains, every four hours, increasing one grain with every dose. Oleate of mercury, 5 per cent., half a drachm inunctions daily.

November 5th. Moderate dilatation and insensibility of pupils for the past three days; nystagmus more marked.

November 6th. Clonic spasm of left arm and fingers, lasting about a minute.

November 7th. Child seems a little less stupid to-day; moves right foot fairly well when raised, and trying to walk, but drags the left one. Oleate of mercury discontinued.

November 11th. Spinal canal aspirated between the third and fourth lumbar vertebræ, and about one drachm of clear, watery fluid withdrawn. An examination by Dr. Brooks failed to disclose any tubercle bacilli. Iodide of potash discontinued.

November 15th. No change in the child's condition since the aspiration until to-day, when a slight retraction of the muscles of the neck was noticed. She coughs occasionally.

November 17th. Child does not reply when spoken to, but nods its head.

November 19th. Takes food badly; hard to get the child to swallow. Dr. Davis examined the eyes, and found a papillitis beginning in the right eye, with swelling of the optic disk and tortuosity of the veins. Weight, 15 pounds, 7 ounces.

November 22d. Eyes show no change since the 19th. Rectal feeding is employed on account of the difficulty of swallowing.

November 23d. She will not take any food by mouth.

November 25th. A purulent discharge from the vagina noted.

November 29th. Coarse râles heard over both chests; some exophthalmos present.

December 1st. Fed by stomach tube. Weight, 13 pounds, 3 ounces.

December 3d. Pulse very weak and irregular; respiration also irregular. The child grew weaker, and died December 7th.

Temperature.—The temperature ranged from 98° to 100° F. up to November 26th, the evening temperature being a little higher than the morning—half to one degree. On November 26th it was 101°; on December 2d, 103.4°; December 2d, 99.2°; December 4th, 101.4°; December 6th, 102.4°.

Pulse.—Varied from 70 to 140.

Respirations.—The respirations also varied. The lowest at any time was 12, and the highest, 60 per minute. The rhythm of respiration was irregular throughout the stay in the hospital, though the number of respirations per minute averaged between 16 and 26.

Autopsy.—Miliary tubercles were found scattered through the lungs, and the bronchial glands were enlarged and tuberculous. The left apex was solid. The brain was carefully examined by Dr. Collins, and showed three distinct tumors about the size of walnuts, viz., (1) on the basal surface of the left cerebellar lobe; (2) at the head of the caudate nucleus on the right side; and (3) in the substance of the anterior and posterior quadrigeminal bodies. The tumor on the under surface of the cerebellum seemed to be attached only to the pia. The nature of the growths was tuberculous.

On admission to the hospital, and after examination, the diagnosis of tumor of the brain, either tubercular or syphilitic, was made, but the tumors were not localized. As noted above, no help in the diagnosis was afforded by lumbar puncture. It is extremely difficult to detect tubercle bacilli in the cerebro-spinal fluid. The writer has employed lumbar puncture in a number of cases of tubercular meningitis, and an examination by a competent bacteriologist failed to discover bacilli.

51 WEST FIFTY-FIRST STREET.

NOTES OF A CASE IN WHICH MARKED ENLARGEMENT OF THE LIVER, ASSOCIATED WITH SYMPTOMS RESEMBLING THOSE OF TYPHOID FEVER, OCCURRED IN A YOUNG CHILD.

BY A. D. BLACKADER, M.D.,

Professor of Pharmacology and Therapeutics, and Lecturer on Diseases of Children, McGill University, Montreal.

The usual conditions under which notable enlargement of the liver may occur in children are well recognized, and on its appearance we look for symptoms pointing to the presence of syphilis, tuberculosis, leukæmia, alcoholic or malarial poisoning, or amyloid or cardiac disease. Slight enlargement from congestion may sometimes be due to the absorption of ptomaines from the intestinal tract in cases of faulty digestion; to the administration of food containing articles more or less irritating to the hepatic cells; or to the action of toxins generated in the system during the course of some of the infectious fevers.

In the following case the enlargement was very notable. It began toward the close of the second week of fever, reached its maximum about the fourth week, and then slowly receded. It was associated with no tenderness, no ascites, no symptoms of jaundice. A careful examination, twelve weeks after the commencement of the attack, failed to reveal any undue enlargement of the liver, or irregularity in its borders.

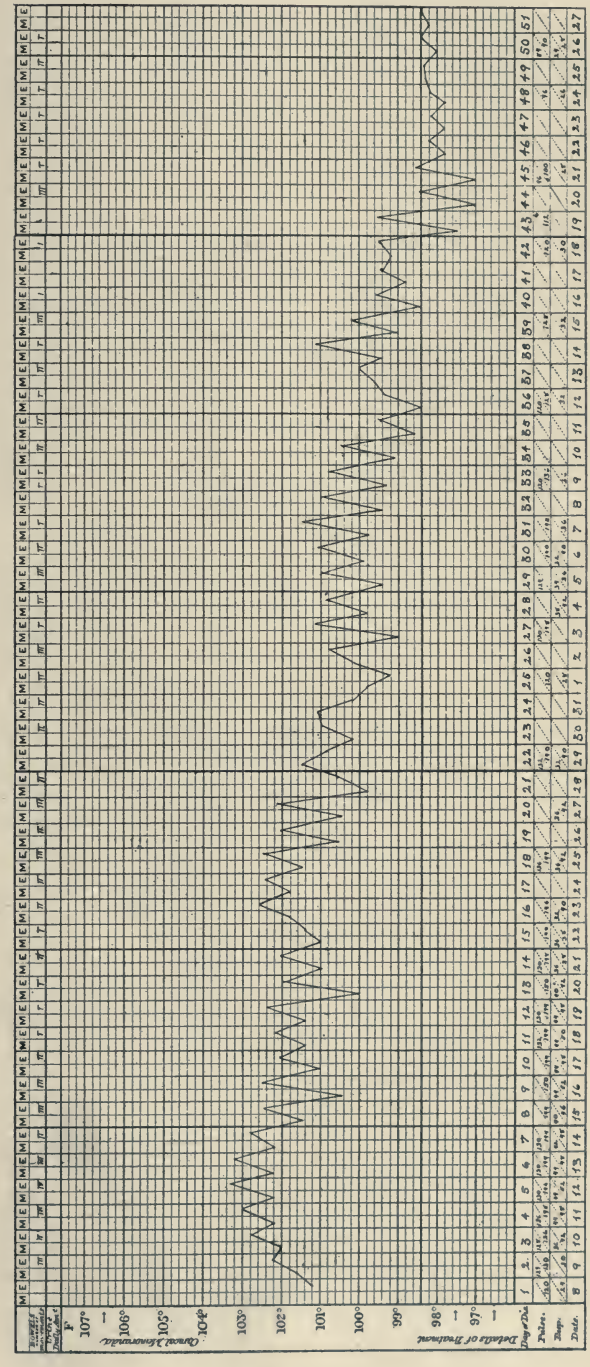
In a very imperfect review of the literature on the subject, I have failed to note any reference made to a similar condition, with the exception of some cases reported recently in the *Journal de Clinique et de Thérapeutique Infantiles,* Paris, April 16, 1896, by Dr. Edouard Tordeus, of Brussels. This writer gives the history of five cases of lobar pneumonia, in which a very notable, but temporary, enlargement of the liver made its appearance a few days after complete defervescence had taken place. In two of these the firm, smooth edge could be distinctly felt as low down as the level of the umbilicus. There was no tenderness on palpation, no icterus, no ascites. The spleen was not enlarged, the appetite remained excellent, and the patient was in

good spirits. Under suitable regimen the enlargement disappeared in a few weeks.

In my own case, although some of the symptoms simulated those of pneumonia, at no time was I able to obtain, by physical examination, definite symptoms of any consolidation, and the continued pyrexia appeared to oppose any such hypothesis.

The history of the case was as follows:

On the evening of March 8, 1896, I was summoned to see F. S., a bright, precocious child, aged two and a half years, whom I had attended at intervals from a few weeks after birth. Her parents were both in good health. The paternal grandfather died from interstitial nephritis of gouty origin; the father is a dyspeptic, and suffers from neuralgic attacks, probably also of gouty origin. The mother is of a nervous temperament, but is otherwise well. The child herself was nursed by the mother till the end of the third month, when, under my directions, she was gradually weaned. Artificial feeding proceeded very satisfactorily. The child, weighed every week, showed a steady gain in weight. Dentition was normal, and so far she had escaped all the eruptive fevers. Six weeks previously she had suffered from a slight influenzal attack, which had left her looking pale, and for which at the time I had prescribed a ferruginous tonic. On the evening of my visit I found her with flushed face and slightly coated tongue; pulse, 120; respiration, 24; temperature, 101⅘. There was no complaint of pain, nor did a thorough examination reveal any abnormal condition. A simple alkaline mixture was prescribed. The temperature rose slowly, and on the evening of the fourth day reached 103° F. Respirations were now decidedly quickened, and the child appeared to have occasional attacks of pain, but the site of this was obscure, and appeared to be variable. The nights were restless. On the sixth day respiration still remained quickened. There was slight diarrhœa, associated with some pain, and greenish colored stools containing more than a normal amount of mucus. The abdomen was slightly distended, but no special tenderness was elicited on pressure. Splenic dulness was present. No abnormal physical signs were detected after repeated examinations of the chest. No rose spots were visible on the body. On the evening of the eighth day, as the diagnosis was still uncertain, Dr. Browne saw her with me in consultation. The daily range of temperature was now between 102° and 103° F.; the respira-



TEMPERATURE RANGE IN A CASE IN WHICH MARKED ENLARGEMENT OF THE LIVER WAS ASSOCIATED WITH SYMPTOMS RESEMBLING THOSE OF TYPHOID FEVER, IN A YOUNG CHILD.

tions from 40 to 48; the pulse was weak, 140 to 150. Diarrhœa still continued, with about the same characters. Cough could hardly be said to be present. Beyond an occasional sibilant rale no abnormal physical signs were detected in the chest. We both considered the case as possibly one of pneumonia, but no absolute diagnosis was made.

During the following week the symptoms remained about the same. The respirations were less rapid, averaging about 40 per minute, while the pulse varied from 120 to 140. There were two or three relaxed motions per day, of light color, and occasionally associated with some colicky pain. A cough occurred only occasionally, and was very slight in character. Repeated physical examinations revealed only a few loose bronchial râles toward the base. On the evening of March 22d, the area of liver dulness was first observed to be slightly increased, extending a full inch below the margin of the ribs in the nipple line. Splenic dulness increased, but the spleen was not palpable. The abdomen was slightly distended, but no special tenderness was detected on deep pressure.

A specimen of urine was not obtainable without using the catheter, but, from the appearance of the diapers, there was nothing abnormal either in its amount or its character.

The enlargement of the liver gradually increased until, by the 1st of April, its margin was distinctly palpable half an inch below the level of the umbilicus. Its surface and margin were quite smooth. No local tenderness was elicited. The enlargement was general. The lower margin of the spleen was also easily felt, extending one inch below the margin of the ribs. There was no icterus, and no ascites. Slight pitting was perceptible on deep pressure over the tibiœ.

Dr. Lafleur kindly saw the case at this time along with me, and corroborated the physical signs I have already noted. An examination of the blood, at my request, were made by him at this visit. The red corpuscles was found to number 4,666.400 per cubic mm. The white corpuscles 4,600 per cubic mm. There was no poikilocytosis. The red cells were of equal size and well formed. The hæmoglobin was not estimated.

The physical signs persisted for another fortnight, when a gradual diminution in the size of the liver followed a distinct improvement in the general symptoms. Not until the end of the sixth week did complete defervescence take place, and could

convalescence be said to be established. The edge of the liver, at this time, was still distinctly perceptible more than two fingers' breadth below the level of the ribs. It was six weeks later before I could assure the mother that it had regained its normal size.

The question of diagnosis in this case must, it seems to me, still remain obscure; with the probabilities pointing in favor of typhoid fever. The enlargement of the liver is, however, interesting, whatever be the diagnosis, and the future history of such a case will be instructive. That it was an enlargement and not a displacement, may be considered as well established as repeated examinations, verified by two other physicians, could render it. Its origin and character must remain in doubt.

236 MOUNTAIN STREET.

DISCUSSION.

DR. HENRI LAFLEUR.—There was only one examination of the blood made, and the idea of acute leukæmia could be excluded. The total number of leucocytes was 4,600 to the c.c. As regards the rest of the condition I corroborate what Dr. Blackader says.

DR. AUGUST SEIBERT.—I would call attention to the report of Czerny and Moser, assistants of Prof. Epstein, of Prague, published about a year ago in regard to blood examinations in infants suffering from intestinal catarrh. They examined, I think, fifteen cases, and in each case found bacteria in the blood that are otherwise found in the intestines. In most of these cases secondary swelling of the liver, of the spleen, a slight nephritis and broncho-pneumonia were present. Enlargement of the liver and of the spleen, in cases of chronic intestinal catarrh, and sometimes following mild acute intestinal infections, I have seen quite often, and within the last five to eight years have looked upon them as general infections entering the body through the intestine. This case of Dr. Blackader's might have developed in a similar way.

American Pediatric Society
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